ST. FRANCIS HOSPITAL NUMBER

DELAWARE STATE MEDICAL JOURNAL

Official Organ of the Medical Society of Delaware

INCORPORATED 1789

VOLUME 28
NUMBER 11

NOVEMBER, 1956

Per Year, $4.00
Per Copy, $ .50

CONTENTS

APLASIA OF RIGHT LIVER LOBE WITH CHOLELITHIASIS, *Edward M. Bohan, M.D.* 291

THE PRESENT STATUS OF THE TREATMENT OF RHEUMATIC DISEASES, *Philip R. Trommer, M.D.* 293

ASPECTS OF ALCOHOL INTOXICATION, *Frederick Knecht, Jr., LL.B.* 298

THE CASE FOR CLINICAL RESEARCH, *Edward M. Bohan, M.D.* 311

PROCEEDINGS OF HOUSE OF DELEGATES 313

EDITORIAL 325

Entered as second-class matter June 28, 1929, at the Post Office at Wilmington, Delaware, under the Act of March 3, 1879, Editorial and Business Office, 621 Delaware Ave., Wilmington 1, Delaware. Issued monthly. Copyright, 1956 by the Medical Society of Delaware.

Makes your therapy safe and pleasant as well as effective

ILOTYCIN

(Erythromycin, Lilly)

successfully attacks the pathogens you commonly encounter but avoids serious and disturbing side-effects.

Tablets, pediatric suspensions, drops, I.M. and I.V. ampoules.

Lilly

QUALITY / RESEARCH / INTEGRITY

you can count on cooperation when you use.

When you prescribe SUSPENSION CHLOROMYCETIN PALMITATE for sick youngsters, no tears or tantrums at medicine time threaten your dosage schedule. Children readily accept this tempting, custard-flavored preparation of CHLOROMYCETIN (chloramphenicol, Parke-Davis). Succeeding doses are taken as readily as the first, because SUSPENSION CHLOROMYCETIN PALMITATE is easy to swallow and leaves no unpleasant aftertaste.

To simplify therapy still further, SUSPENSION CHLOROMYCETIN PALMITATE does not require refrigeration and may be kept conveniently in the sickroom. Its liquid form enables flexibility of dosage easily.

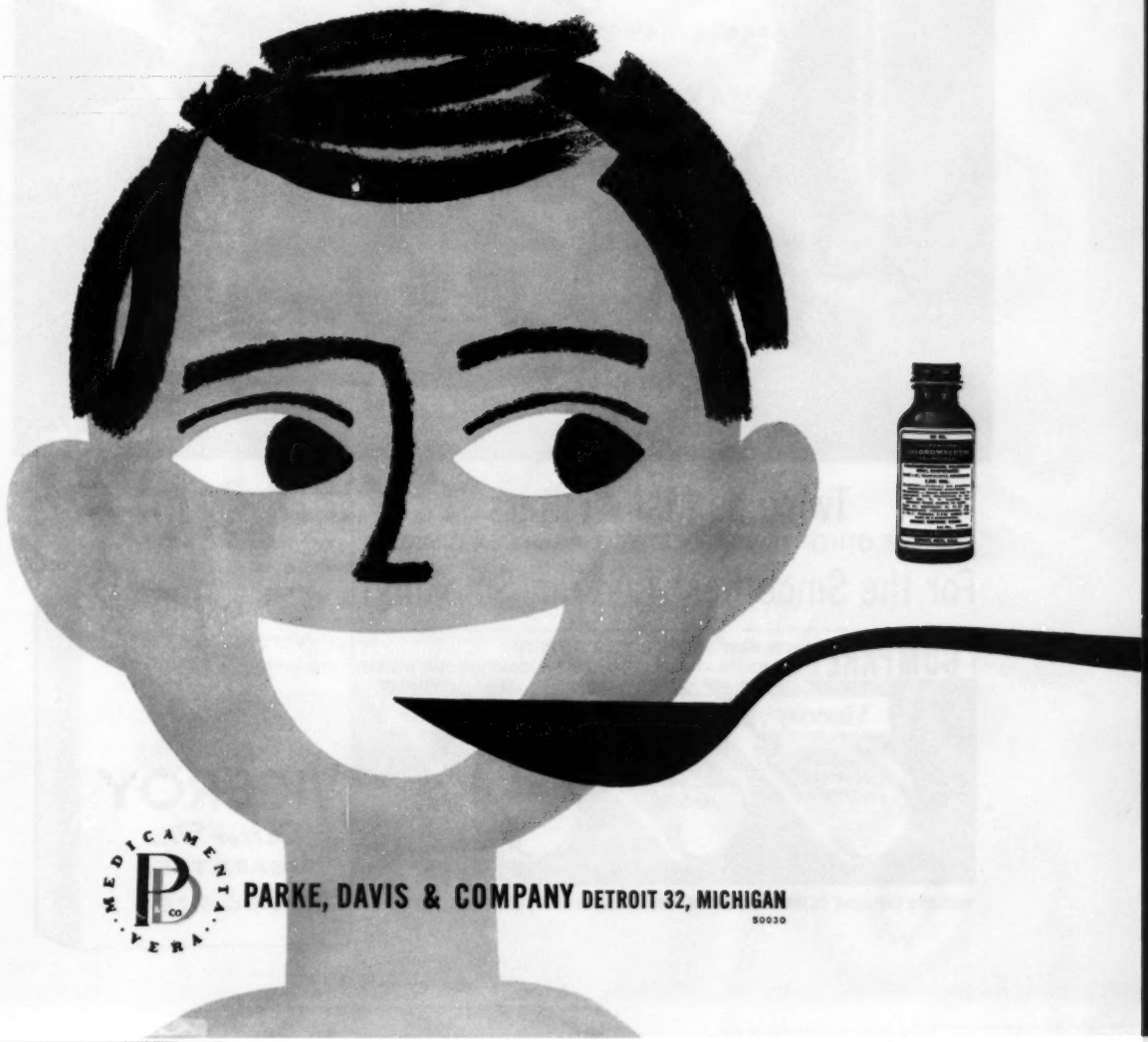
CHLOROMYCETIN is a potent therapeutic agent and, because certain blood dyscrasias have been associated with its administration, it should not be used indiscriminately or for minor infections. Furthermore, as with certain other drugs, adequate blood studies should be made when the patient requires prolonged or intermittent therapy.

. Suspension

Chloromycetin®
Palmitate

pleasant-tasting Chloromycetin for pediatric use

Supplied: SUSPENSION CHLOROMYCETIN PALMITATE, containing the equivalent of 125 mg. of CHLOROMYCETIN per 4 cc., is available in 60-cc. vials.



MEDICAMENTA VERA · PD co

PARKE, DAVIS & COMPANY DETROIT 32, MICHIGAN

50030

HERE'S WHY SO MANY DOCTORS
NOW SMOKE AND RECOMMEND

VICEROY

Twice as Many Filters
AS THE OTHER TWO LARGEST-SELLING FILTER BRANDS
For the Smoothest Taste in Smoking!

COMPARE! HOW MANY FILTERS IN YOUR FILTER TIP?
(REMEMBER—THE MORE FILTERS THE SMOOTHER THE TASTE!)
Viceroy
Brand B
Brand C
TWICE AS MANY FILTERS
Only HALF the FILTERS
LESS than HALF the FILTERS

VICEROY'S EXCLUSIVE FILTER IS MADE FROM PURE CELLULOSE—SOFT, SNOW-WHITE, NATURAL!

relaxes both the body and the mind

well suited for prolonged therapy

- well tolerated, nonaddictive, essentially nontoxic
- no blood dyscrasias, liver toxicity, Parkinson-like syndrome or nasal stuffiness
- chemically unrelated to chlorpromazine or reserpine
- does not produce significant depression
- orally effective within 30 minutes for a period of 6 hours

Indications: **anxiety and tension states, muscle spasm.**

THE ORIGINAL MEPROBAMATE

Miltown®

Tranquilizer with muscle-relaxant action

DISCOVERED AND INTRODUCED

BY WALLACE LABORATORIES, *New Brunswick, N.J.*

2-methyl-2-n-propyl-1,3-propanediol dicarbamate—U.S. Patent 2,724,720

SUPPLIED: *400 mg. scored tablets. Usual dose: 1 or 2 tablets t.i.d.*

Literature and Samples Available on Request

THE MILTOWN MOLECULE

CM 3706 R

NEW

For Pain-Free

of everyday

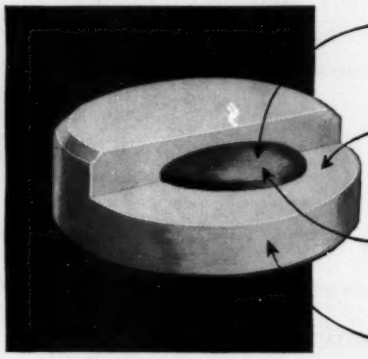
In "Rheumatism"*

Multiple

TEMP

combine:

THE PROPER FORMULA
PROPERLY FORMULATED



Physical separation of the steroid component from the aluminum hydroxide as provided by the Multiple Compressed Tablet construction assures full potency and stability of prednisolone.

PREDNISOLONE (*1 mg.*)

+

ASPIRIN (*0.3 Gm.*).....

+

ASCORBIC ACID (*50 mg.*

+

ANTACID (*0.2 Gm.*).....

*Early rheumatoid arthritis
Rheumatoid spondylitis
Osteoarthritis
Still's disease
Psoriatic arthritis
Bursitis
Synovitis
Tenosynovitis
Myositis
Fibrositis
Neuritis

Performance

activities

Patients

Compressed Tablets

OGEN®

..... for anti-inflammatory, anti-rheumatic benefits *at effective low dosage.*

..... for analgesia plus additional anti-rheumatic activity.

..... for anti-stress support that guards against adrenal ascorbic acid depletion.
(Ascorbic Acid present as 60 mg. Sodium Ascorbate.)

..... dried aluminum hydroxide gel minimizes the possibility of gastric distress.

DOSAGE: *1-4 TEMPOGEN Tablets t.i.d. or q.i.d. (TEMPOGEN Forte, 1 or 2 tablets t.i.d. or q.i.d.) for one or two weeks. Then lower by 1 tablet every four or five days to maintenance level.*

SUPPLIED: *TEMPOGEN and TEMPOGEN Forte —in bottles of 100 Multiple Compressed Tablets. (TEMPOGEN Forte provides 2 mg. of prednisolone.)*

MSD

MERCK SHARP & DOHME
DIVISION OF MERCK & CO., INC.
PHILADELPHIA 1, PA.

little

How to win friends ...

The Best Tasting
Aspirin you can prescribe.

The Flavor Remains Stable
down to the last tablet.

25¢ Bottle of 48 tablets (1¼ grs. each).

We will be pleased to send samples on request.

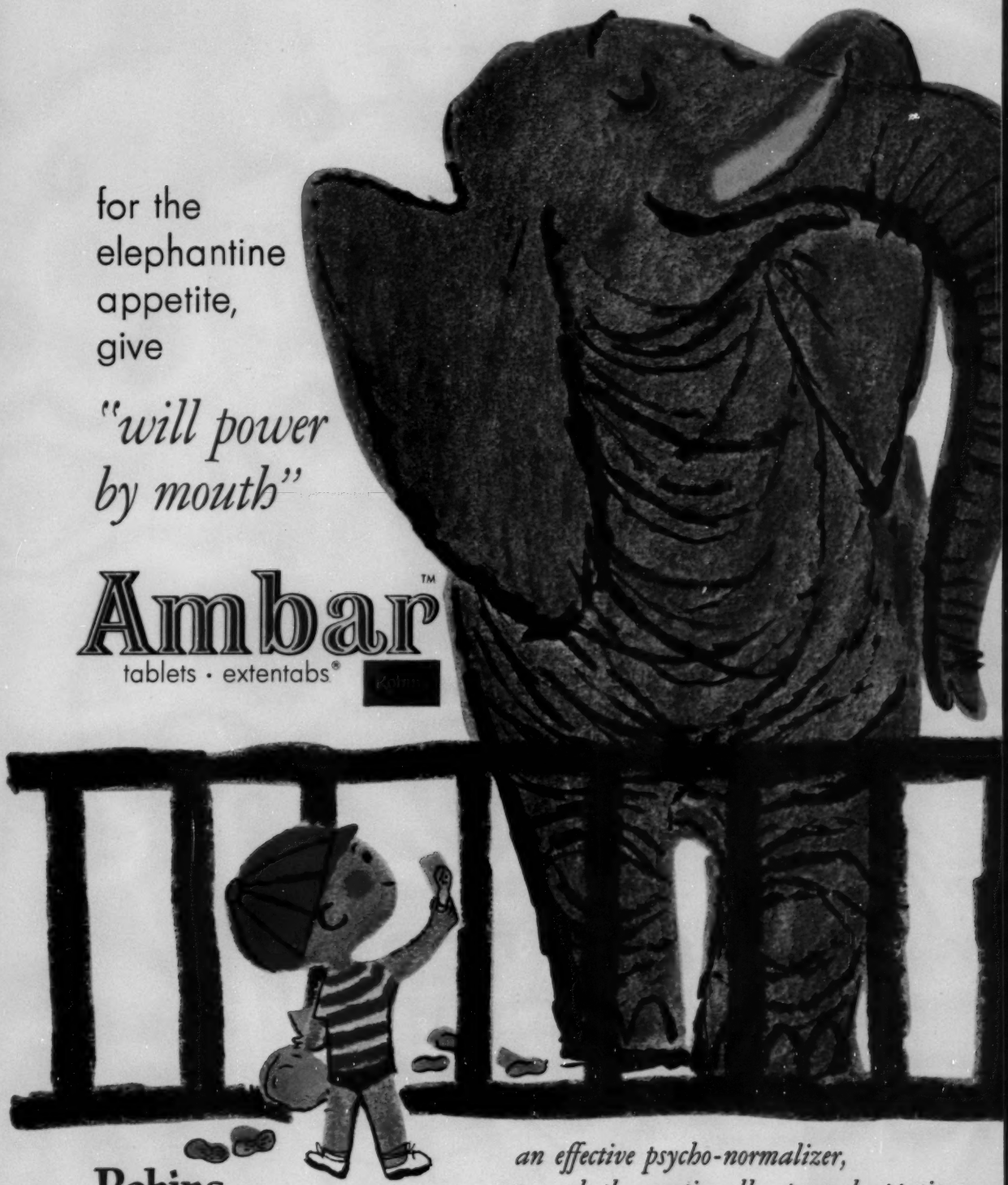
THE BAYER COMPANY DIVISION
of Sterling Drug Inc.
1450 Broadway, New York 18, N. Y.

for the elephantine appetite, give

"will power by mouth"

Ambar™

tablets • extentabs®



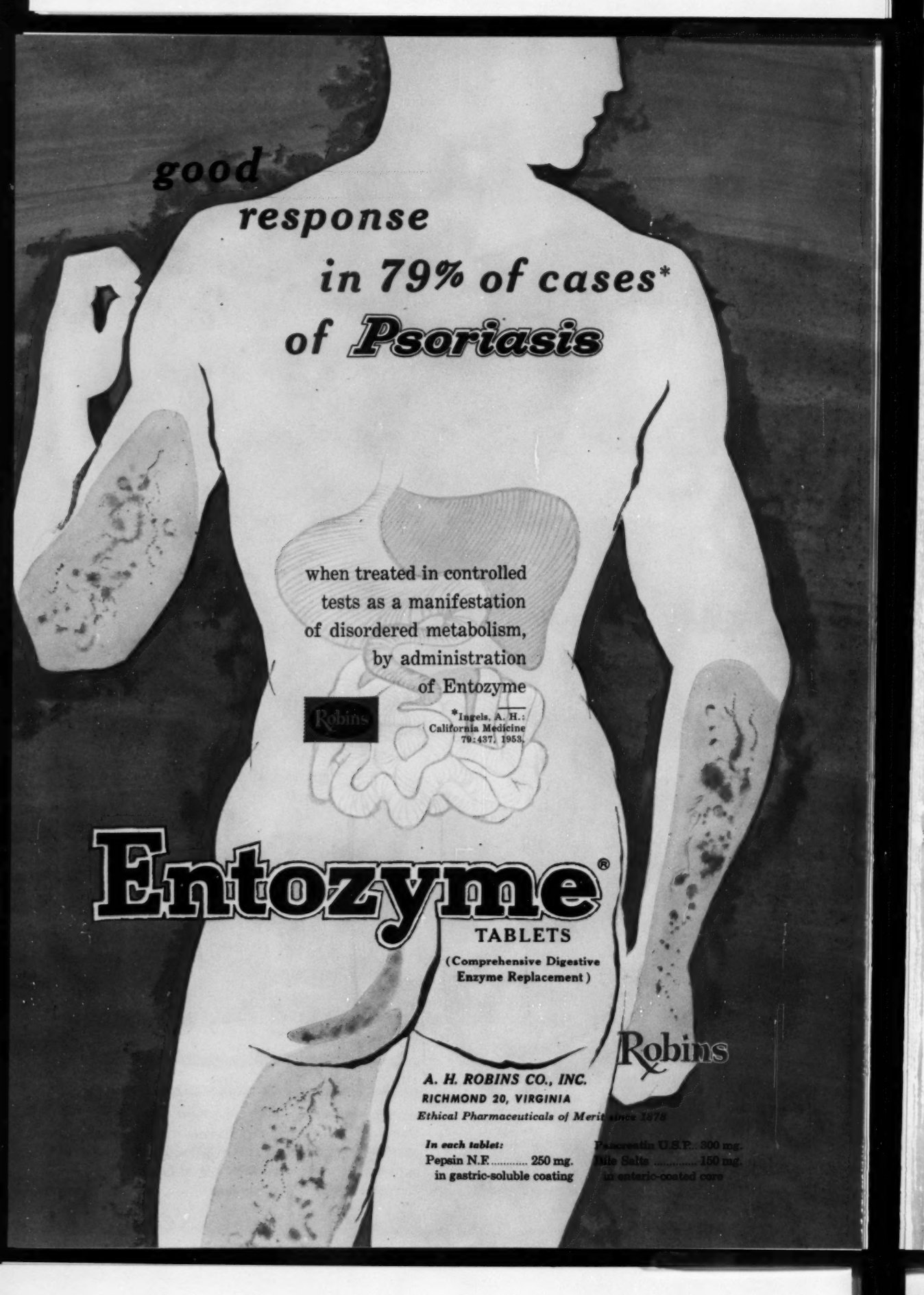
Robins

	In each *Tablet*	*Extentab*
Methamphetamine	3.33 mg.	10.0 mg.
Phenobarbital	21.6 mg. (⅓ gr.)	64.8 mg. (1 gr.)

A. H. ROBINS CO., INC., Richmond 20, Virginia
Ethical Pharmaceuticals of Merit since 1878

an effective psycho-normalizer, to curb the emotionally spurred appetite

Methamphetamine for central nervous stimulation (more potent than amphetamine) • with phenobarbital control, in optimal ratio • as plain Tablets — or as Extentabs®, that work all-day on a single dose


good
response
in 79% of cases*
of Psoriasis
when treated in controlled
tests as a manifestation
of disordered metabolism,
by administration
of Entozyme
Robins
*Ingels, A. H.:
California Medicine
79:437, 1953.
Entozyme®
TABLETS
(Comprehensive Digestive
Enzyme Replacement)
Robins
A. H. ROBINS CO., INC.
RICHMOND 20, VIRGINIA
Ethical Pharmaceuticals of Merit since 1878
In each tablet:
Pepsin N.F..... 250 mg.
in gastric-soluble coating
Pancreatin U.S.P.. 300 mg.
Bile Salts ..... 150 mg.
in enteric-coated core

Lysine plus vitamins

to help children eat more, grow more!

INCREMIN combines the amino acid lysine with vitamins B_1, B_6 and B_{12}—essential nutrients that stimulate appetite, and promote more efficient utilization of protein. For children who are problem eaters, for the underweight, for the generally below-normal child—INCREMIN will usually produce a remarkable and prompt improvement!

Cherry flavor. Can be mixed with milk, milk formula, or other liquid. In 15 cc. polyethylene dropper bottle.

Dosage: 0.5 to 1 cc. (10-20 drops) daily. Each cc. (20 drops) contains:

l-Lysine HCl	300 mg.
Vitamin B_{12}	25 mcgm.
Thiamine HCl (B_1)	10 mg.
Pyridoxine HCl (B_6)	5 mg.
Alcohol	1%

Excellent for the elderly! INCREMIN serves equally well to stimulate lagging appetites in geriatric patients.

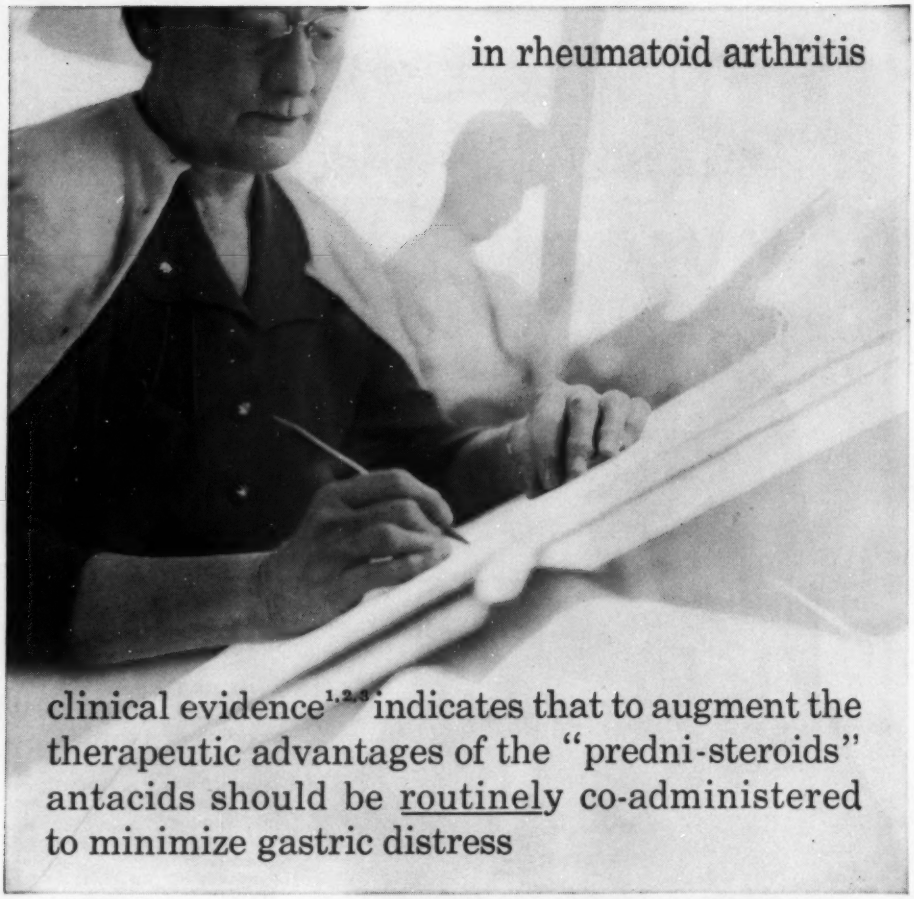
New

INCREMIN*

Lysine-Vitamin Drops

Lederle LEDERLE LABORATORIES DIVISION, AMERICAN CYANAMID COMPANY, PEARL RIVER, NEW YORK

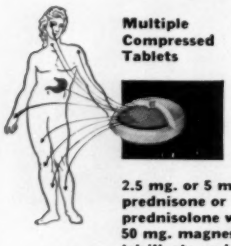
*REG. U.S. PAT. OFF.



clinical evidence[1,2,3] indicates that to augment the therapeutic advantages of the "predni-steroids" antacids should be routinely co-administered to minimize gastric distress

ROUTINE CO-ADMINISTRATION MEANS

'Co-Hydeltra'
(Prednisolone Buffered)



Multiple Compressed Tablets

2.5 mg. or 5 mg. prednisone or prednisolone with 50 mg. magnesium trisilicate and 300 mg. aluminum hydroxide gel.

'Co-Deltra'
(Prednisone Buffered)

All the benefits of the "predni-steroids" plus positive antacid action to minimize gastric distress.

References: 1. Boland, E. W., *J.A.M.A.* **160**:613 (February 25) 1956. 2. Margolis, H. M. *et al.*, *J.A.M.A.* **158**:454 (June 11) 1955. 3. Bollet, A. J. *et al.*, *J.A.M.A.* **158**:459 (June 11) 1955.

MSD
MERCK SHARP & DOHME
DIVISION OF MERCK & CO., INC.
PHILADELPHIA 1, PA.

'CO-DELTRA' *and* 'CO-HYDELTRA' *are trademarks of* MERCK & CO., INC.

Journal Report:

Hypertensive symptoms relieved in 96% of patients

"Comparison of pentolinium [ANSOLYSEN] with other preparations in 25 patients with severe essential hypertension, for whom all other methods of management had failed, showed that pentolinium is the most effective of available agents in reducing dangerously high blood pressure to the desired levels, and in modifying some of the complications of hypertension, as cardiac decompensation, cardiomegaly and retinopathy. . . .

"In 96 per cent (24 patients) clinical symptoms were relieved and the blood pressure maintained at comfortable levels. . . ."[1]

ANSOLYSEN®

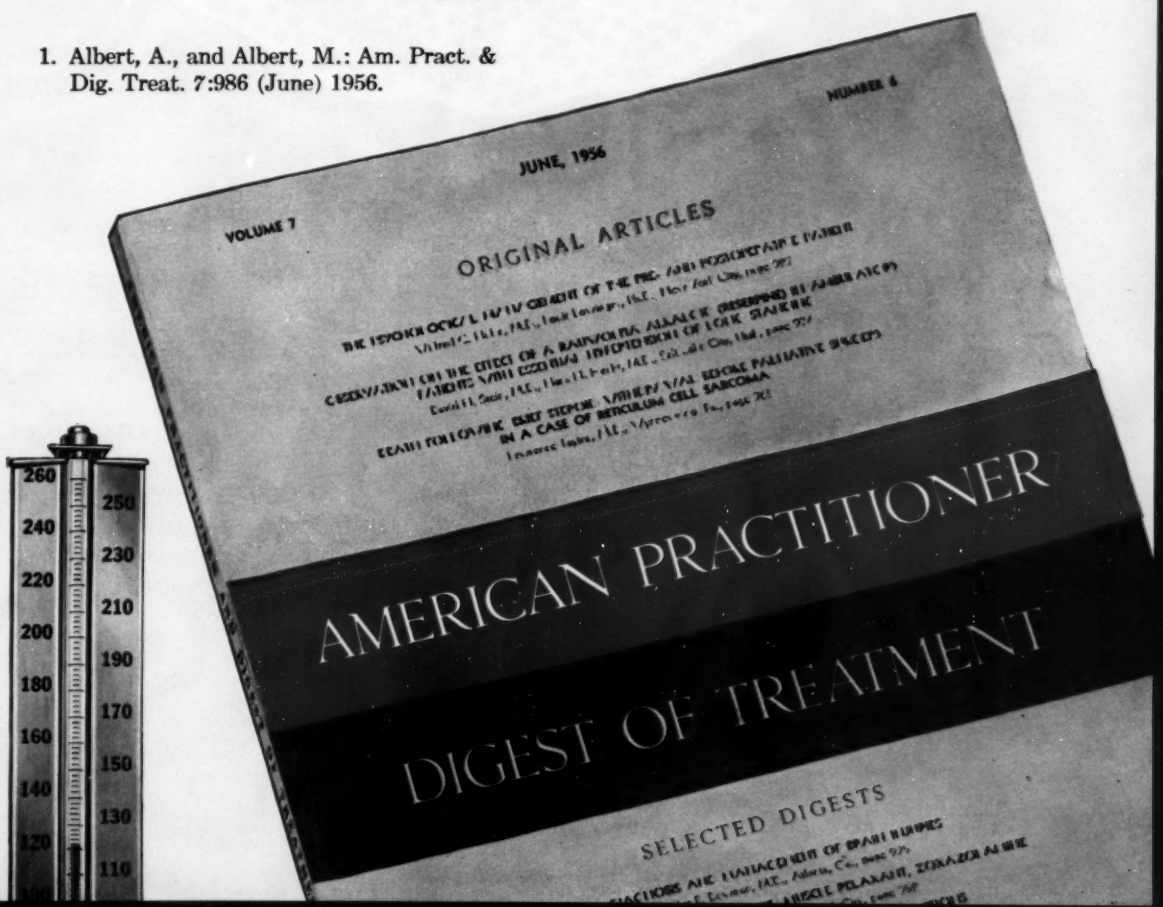
TARTRATE

Pentolinium Tartrate

Wyeth®
Philadelphia 1, Pa.

Lowers Blood Pressure

1. Albert, A., and Albert, M.: Am. Pract. & Dig. Treat. 7:986 (June) 1956.



highly successful . . .

- faster relief of pain, photophobia
- better control of inflammation, edema, allergy
- effective against common eye pathogens
- extremely well tolerated

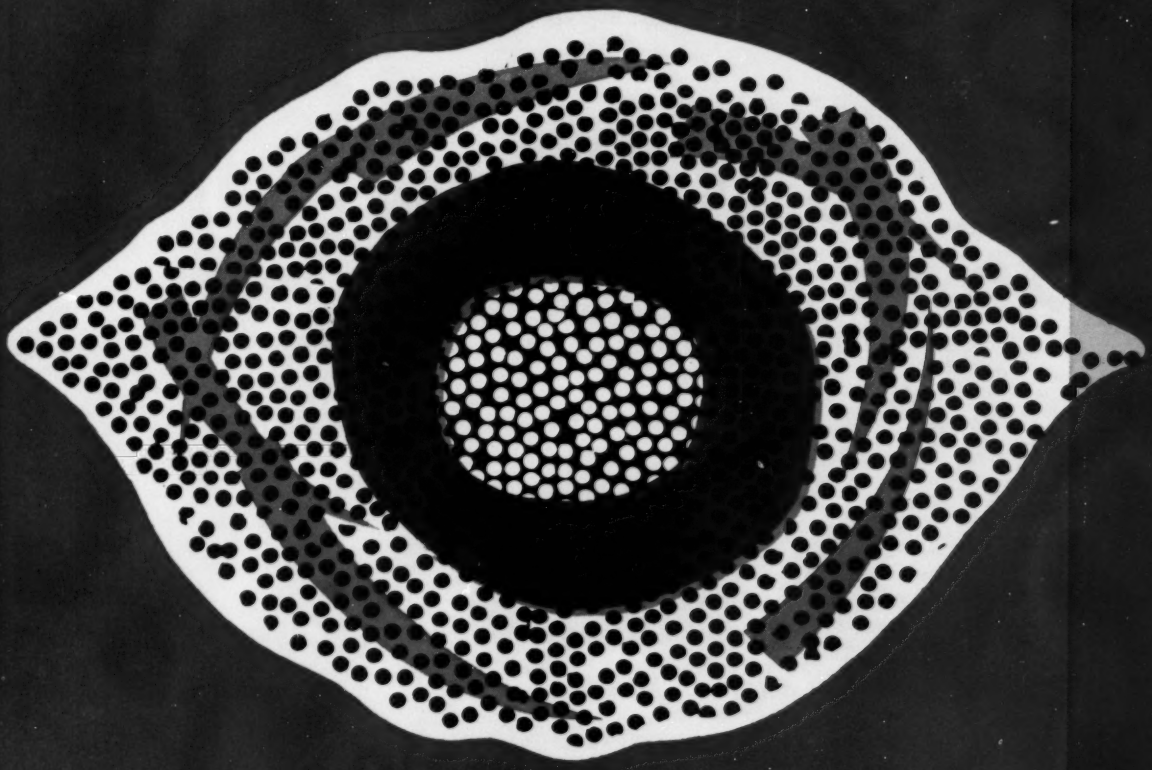
now available

for inflammatory, allergic, infectious or traumatic eye conditions amenable to topical therapy—rapid, potent, topical Meti-steroid and anti-infective action

supplied: METIMYD Ophthalmic Suspension-*Sterile*: prednisolone acetate (METICORTELONE Acetate) 5 mg. per cc. (0.5%) suspended in an isotonic buffered and preserved solution of sulfacetamide sodium 100 mg. per cc. (10%), 5 cc. dropper bottle. METIMYD Ointment with Neomycin: each gram contains 5 mg. prednisolone acetate (METICORTELONE Acetate), 100 mg. sulfacetamide sodium and 2.5 mg. neomycin sulfate (equivalent to 1.75 mg. neomycin base); ⅛ oz. tube, boxes of 1 and 12.

METIMYD,® brand of prednisolone acetate and sulfacetamide sodium.
METICORTELONE,® brand of prednisolone.
®T.M.

. . . in topical eye therapy



new

METIMYD*

(prednisolone acetate and sulfacetamide sodium)

Ophthalmic Suspension-Sterile

and

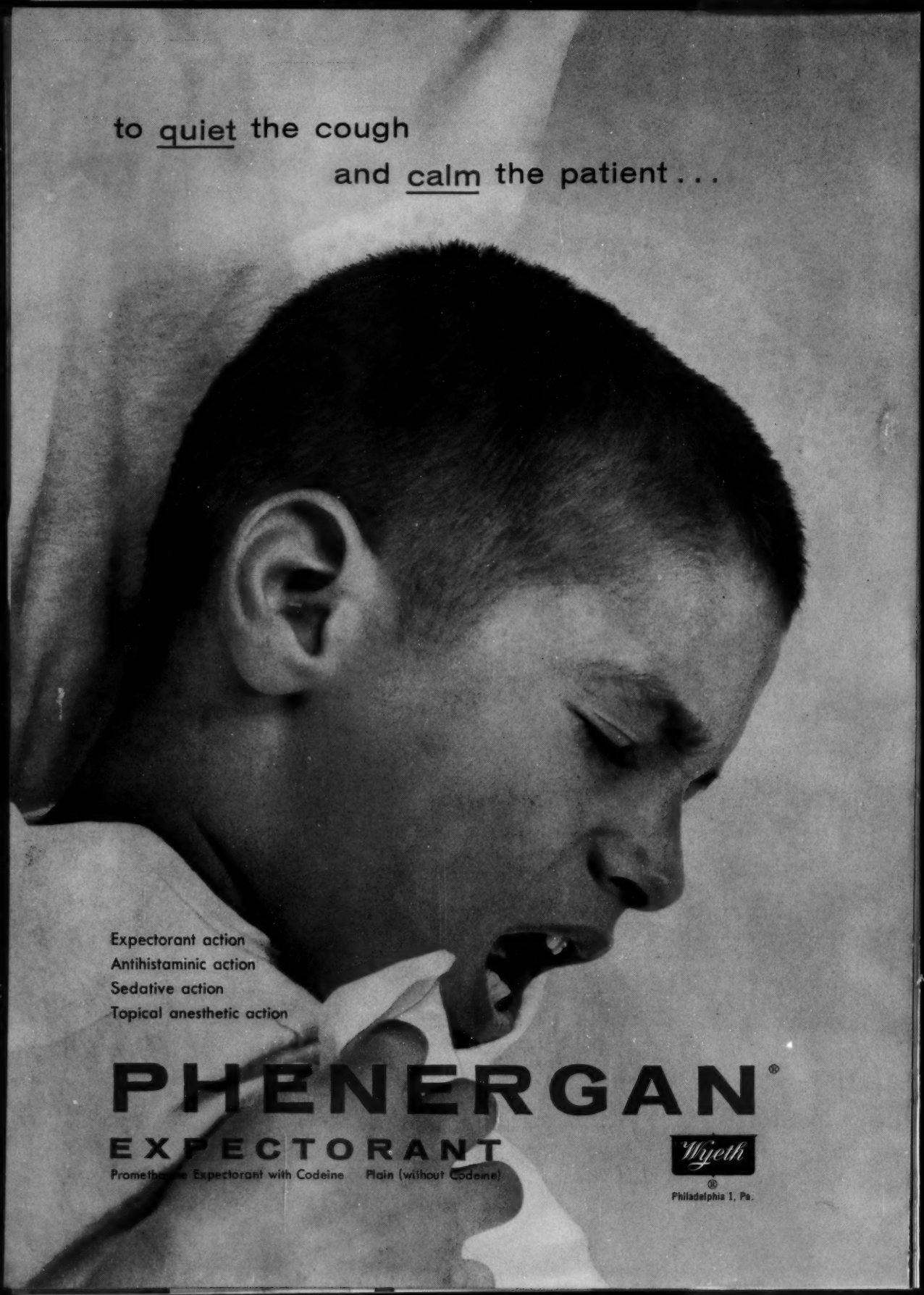
METIMYD

(prednisolone acetate and sulfacetamide sodium with neomycin sulfate)

Ointment with Neomycin

• antibacterial • antiallergic • anti-inflammatory

Schering


to quiet the cough
and calm the patient . . .
Expectorant action
Antihistaminic action
Sedative action
Topical anesthetic action
PHENERGAN®
EXPECTORANT
Promethazine Expectorant with Codeine Plain (without Codeine)
Wyeth®
Philadelphia 1, Pa.

Sodium Restriction

A. Indications

1. Congestive heart failure
2. Hypertension
3. Cirrhosis
4. Pregnancy edema
5. Obesity

B. Management

1. Salt Free Diet
2. Salt Substitutes

℞

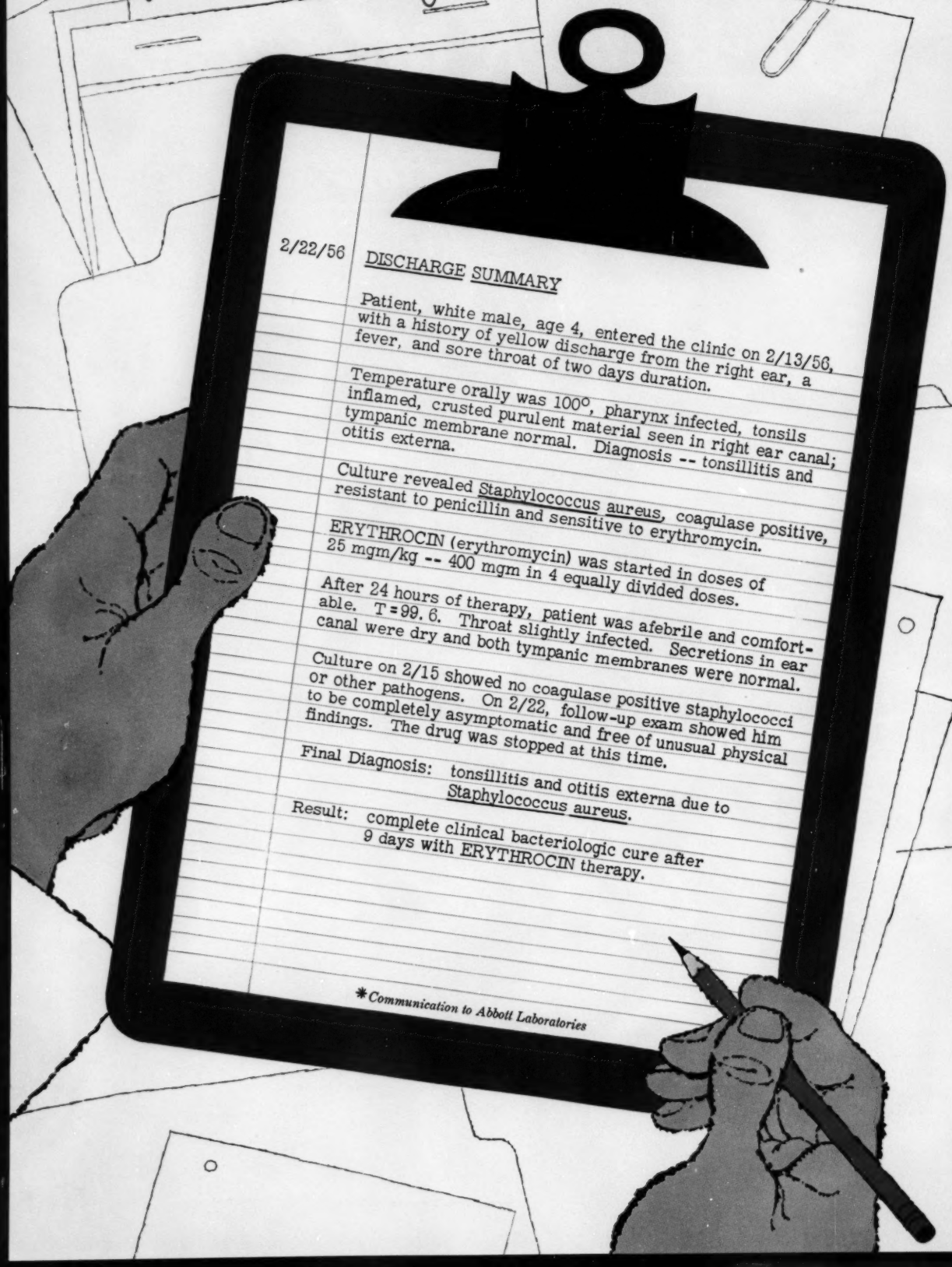
Neocurtasal

New improved formula – now the best tasting. Used like table salt.

Winthrop

Neocurtasal ®
2 oz. Shakers + 8 oz. bottles

Erythrocin in treating tonsillitis and otitis externa*



2/22/56

DISCHARGE SUMMARY

Patient, white male, age 4, entered the clinic on 2/13/56, with a history of yellow discharge from the right ear, a fever, and sore throat of two days duration.

Temperature orally was 100°, pharynx infected, tonsils inflamed, crusted purulent material seen in right ear canal; tympanic membrane normal. Diagnosis -- tonsillitis and otitis externa.

Culture revealed Staphylococcus aureus, coagulase positive, resistant to penicillin and sensitive to erythromycin.

ERYTHROCIN (erythromycin) was started in doses of 25 mgm/kg -- 400 mgm in 4 equally divided doses.

After 24 hours of therapy, patient was afebrile and comfortable. T = 99.6. Throat slightly infected. Secretions in ear canal were dry and both tympanic membranes were normal.

Culture on 2/15 showed no coagulase positive staphylococci or other pathogens. On 2/22, follow-up exam showed him to be completely asymptomatic and free of unusual physical findings. The drug was stopped at this time.

Final Diagnosis: tonsillitis and otitis externa due to Staphylococcus aureus.

Result: complete clinical bacteriologic cure after 9 days with ERYTHROCIN therapy.

*Communication to Abbott Laboratories

2 oz. Shakers + 8 oz. bottles

"clinical response good or excellent"

In one recent study, 18 patients with acute follicular tonsillitis and septic sore throat, were given erythromycin. Infecting organism was *Str. pyogenes*. The investigator stated, *"In all 18, the clinical response could be regarded as either good or excellent."*[1]

This, of course, is only one of many reports showing the effectiveness of ERYTHROCIN against coccic infections. You'll get the same good results (nearly 100% in common, bacterial respiratory infections) when your prescription reads *Filmtab* ERYTHROCIN Stearate.

"toxicity lower in erythromycin-treated patients"

After a study of 208 patients treated with erythromycin (78), procaine penicillin (78) and a placebo (52), the investigator stated: *". . . the incidence of toxicity* (compared to procaine penicillin) *was significantly lower in the erythromycin-treated patients."*[1]

Actually, ERYTHROCIN stands on a remarkable record of safety. After four years, there's not a single report of a severe or fatal reaction attributable to erythromycin. Also, allergic reactions rarely occur. *Filmtab* ERYTHROCIN Stearate (100 and 250 mg.), is available in bottles of 25 and 100, at all pharmacies.

Abbott

® Filmtab—Film sealed tablets, Abbott; pat. applied for.

1. Herrell, W. E., Erythromycin, Antibiotics Monographs, No. 1, p. 29, New York, Medical Encyclopedia, Inc., 1955.
Idem p. 30.

filmtab®

Erythrocin® STEARATE

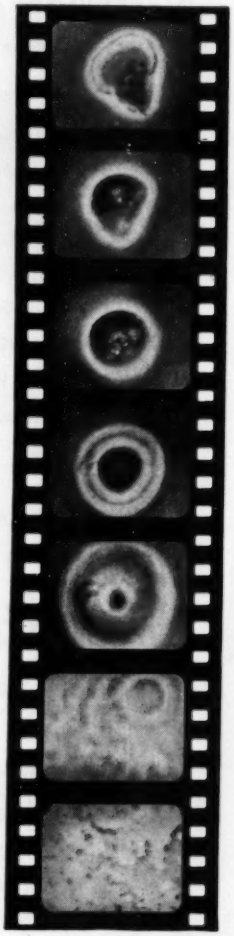
(Erythromycin Stearate, Abbott)

611243

HARD-TO-KILL TRICHOMONADS

EXPLODE

WITHIN 15 SECONDS' CONTACT WITH **VAGISEC** LIQUID



Top to bottom:
2 sec. CONTACTS
4 sec. COMPLEXES
6 sec. DISSOLVES
8 sec. DENATURES
10 sec. SWELLS
15 sec. EXPLODES
16 sec. SCATTERS

WITH THE Davis technique,† using VAGISEC® liquid and jelly, flare-ups of vaginal trichomoniasis rarely occur. VAGISEC liquid actually explodes trichomonads within 15 seconds after douche contact.[1] Better than 90 per cent apparent cures follow use of this new trichomonacide,[2] developed as "Carlendacide" by Dr. Carl Henry Davis, noted gynecologist and author, and C. G. Grand, research physiologist.[3]

No trichomonad escapes—The overwhelming action of VAGISEC liquid dooms the trichomonad. One chelating agent and two surface-acting agents combine in attack to weaken the cell membrane, to remove waxes and lipid materials from the membrane surface, and to denature the protein. With its cell wall destroyed, the parasite imbibes water, swells and explodes. All this occurs within 15 seconds. Only scattered fragments remain.

No other agent or combination of agents kills the trichomonad in this specific fashion or with the *speed* of VAGISEC liquid.[3] When the patient uses VAGISEC jelly as well—the recommended routine—these good effects continue indefinitely.[4]

Reaches hidden trichomonads—Unlike many agents, VAGISEC liquid thoroughly penetrates and dissolves the cellular debris and mucoid material lining the vaginal surface.[3] It reaches hidden trichomonads—often the cause of treatment failure—as well as parasites swimming freely in the canal.

The Davis technique—Office therapy with VAGISEC liquid is combined with home treatment. Both liquid and jelly are prescribed.

OFFICE TREATMENT—*Wipe vaginal walls dry with cotton balls, then wash thoroughly for about three minutes with a 1:100 dilution of* VAGISEC *liquid. Remove excess fluid with cotton balls. Dr. Davis recommends three treatments the first week, two the second and one the third.*

HOME TREATMENT—*Patient douches with* VAGISEC *liquid every night or morning and then inserts* VAGISEC *jelly. Home treatment is continued through two menstrual periods, but omitted on office treatment days. Douching is contraindicated in pregnancy.*

Husband re-infects wife—Since "trichomonads may be passed from the infected male to the uninfected partner during coitus,"[5] prevent re-infection by recommending the use of prophylactics. Specify RAMSES,® the finest possible rubber prophylactic, transparent, very thin yet strong; or XXXX (FOUREX)® skins, of natural animal membrane—pre-moistened. Your prescription of one of these brands insures the protection afforded by Schmid quality prophylactics and assures full acceptance of your regimen. At all pharmacies.

Active ingredients in VAGISEC liquid: Polyoxyethylene nonyl phenol, Sodium ethylene diamine tetra-acetate, Sodium dioctyl sulfosuccinate. In addition, VAGISEC jelly contains Boric acid, Alcohol 5% by weight.

VAGISEC, RAMSES and XXXX (FOUREX) are registered trade-marks of Julius Schmid, Inc.

†Pat. App. for

JULIUS SCHMID, INC.
gynecological division
423 West 55th St., New York 19, N. Y.

References: 1. Davis, C. H.: J.A.M.A. *157*:126 (Jan. 8) 1955. 2. Davis, C. H.: West. J. Surg. *63*:53 (Feb.) 1955. 3. Davis, C. H., and Grand, C. G.: Am. J. Obst. & Gynec. *68*:559 (Aug.) 1954. 4. Davis, C. H. (Ed.): Gynecology and Obstetrics (revision), Hagerstown, Md., W. F. Prior, 1955, vol. 3, chap. 7, pp. 23-33. 5. Draper, J. W.: Internat. Rec. Med. *168*:563 (Sept.) 1955.

Brings
new certainty to
antibiotic therapy
particularly for the
90% of patients
treated in home
or office

new
Sigmamycin*
OLEANDOMYCIN TETRACYCLINE

*TRADEMARK

a new maximum
in therapeutic
effectiveness

a new maximum
in protection
against
resistance

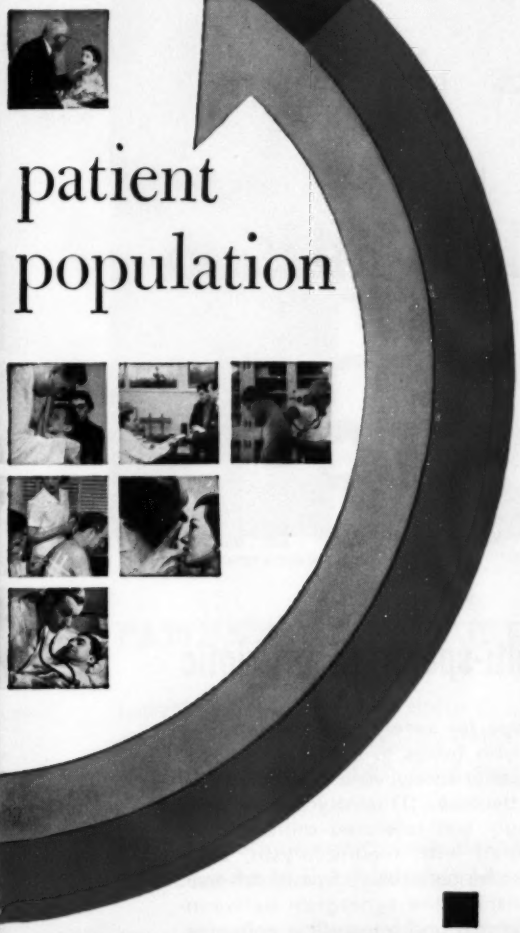
a new maximum
in safety and
toleration

for
your
entire

multi-spectrum
synergistically
strengthened...

Sigma

OLEANDOMYCIN TETRACYCLINE



patient
population

mycin*

a new certainty

in antibiotic therapy, particularly for the 90% of patients treated at home and in the office

Superior control of infectious diseases through superior control of the changing microbial population is now available in a new formulation of tetracycline, outstanding broad-spectrum antibiotic, with oleandomycin, Pfizer-discovered new antimicrobial agent which controls resistant strains. The synergistic combination now brings to antibiotic therapy: (1) a new fuller antimicrobial spectrum which includes even "resistant" staphylococci; (2) new superior protection against emergence of *new* resistant strains; (3) new superior safety and toleration.

*TRADEMARK

Pfizer

superior control
of infectious disease through
superior control of the
changing microbial population

new
Sigmamycin*
OLEANDOMYCIN TETRACYCLINE

A synergistically strengthened multi-spectrum antibiotic

Sigmamycin is a new antibiotic formulation providing: (1) the unsurpassed broad-spectrum activity of tetracycline, the outstanding broad-spectrum antibiotic discovered and identified by Pfizer; (2) the action of oleandomycin, the new antimicrobial agent which combats those strains, particularly among staphylococci, now resistant to tetracycline and other antibiotics.

Sigmamycin embodies a new concept in the use of antibiotics, for with this new synergistically active preparation, the development of refractory pathogens and their emergence as important sources of superinfection are more fully controlled.

New superior safety and toleration— Sigmamycin brings to antibiotic therapy new superior safety, new unexcelled toleration because: (1) tetracycline, an outstandingly well-tolerated antibiotic, is formulated with oleandomycin, also known to be remarkably free of adverse reactions; (2) the synergism between oleandomycin and tetracycline enhances antimicrobial potency.

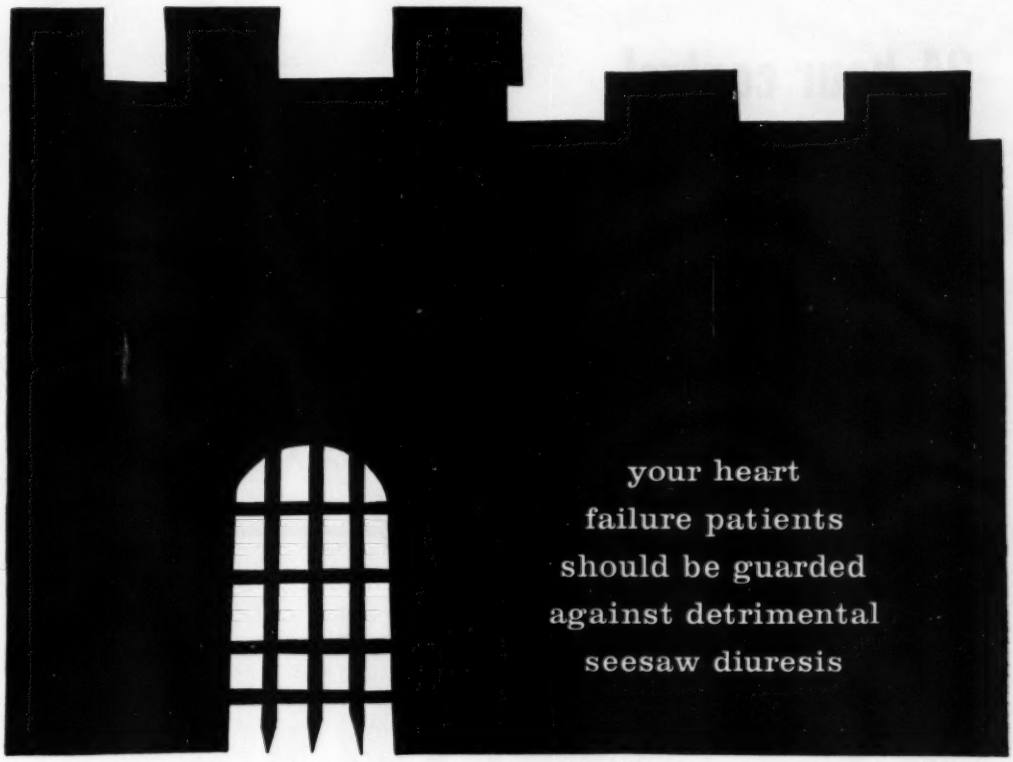
Dosage: 1 to 2 capsules q.i.d.

Supplied: Capsules, 250 mg. (oleandomycin 83 mg., tetracycline 167 mg.) Bottles of 16 and 100.

*TRADEMARK

Pfizer

PFIZER LABORATORIES, *Division, Chas. Pfizer & Co., Inc.*, Brooklyn 6, N. Y.



PATIENTS IN FAILURE NEED AN ORGANOMERCURIAL

Limiting dosage to once daily to avoid refractoriness, or omitting alternate days to circumvent gastrointestinal irritation—necessary with some diuretics—results in a seesaw of diuresis with fluid reaccumulation and recurrent strain on the already failing heart.

With the organomercurials, dosage is individualized and administered as needed, to produce sustained, dependable diuresis.

TABLET

NEOHYDRIN®

BRAND OF CHLORMERODRIN (18.3 MG. OF 3-CHLOROMERCURI-2-METHOXY-PROPYLUREA EQUIVALENT TO 10 MG. OF NON-IONIC MERCURY IN EACH TABLET)

a standard for initial control of severe failure

MERCUHYDRIN® SODIUM

BRAND OF MERALLURIDE INJECTION

LAKESIDE

02356

24-hour control

for the majority of diabetics

GLOBIN INSULIN

'B.W. & CO.'®

a clear solution . . . easy to measure accurately

Discovered by Reiner, Searle, and Lang
in The Wellcome Research Laboratories

BURROUGHS WELLCOME & CO. (U.S.A.) INC. • Tuckahoe 7, New York

Upjohn

Rheumatoid arthritis, rheumatic fever, intractable asthma, allergies...

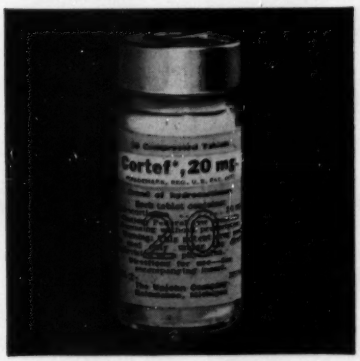
Cortef* *tablets*

Supplied:

5 mg. tablets in bottles of 50
10 mg. tablets in bottles of 25, 100, 500
20 mg. tablets in bottles of 25, 100, 500

*REGISTERED TRADEMARK FOR THE UPJOHN BRAND OF HYDROCORTISONE (COMPOUND F)

The Upjohn Company, Kalamazoo, Michigan



when dandruff stands out as a sign

prescribe **SEBIZON** Lotion

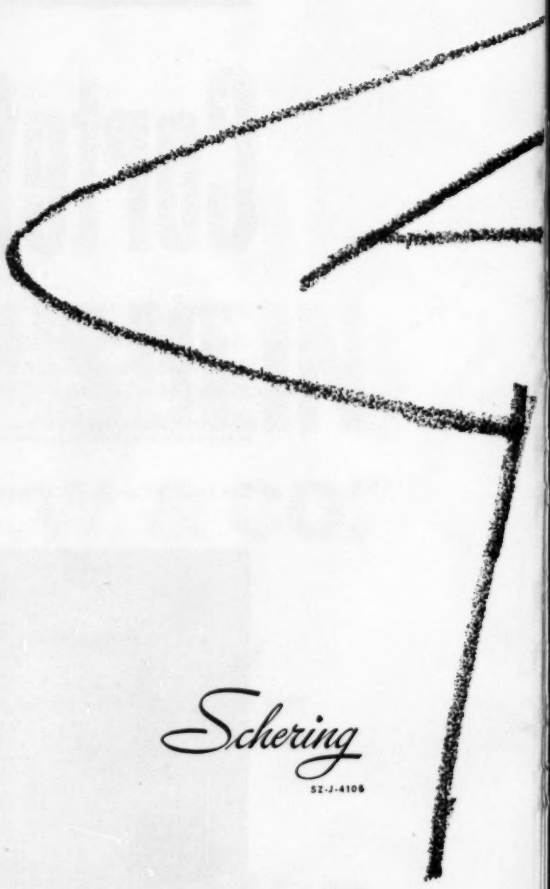
for an extra therapeutic dividend

a method of choice for **rapid control** of seborrhea of the scalp and seborrheic dermatitis in children as well as adults...**no complicated shampoo or timing procedures:** patient rubs in SEBIZON any time of the day, washes out when convenient ...acts as hair dressing: **no odor, no oily or greasy residue,** no tinting of hair.

especially useful when dandruff escapes control again

antiseborrheic and anti-infective
SEBIZON is a cream-type vanishing lotion containing 10% sulfacetamide sodium.

available on prescription only in 3 oz. plastic squeeze tube.



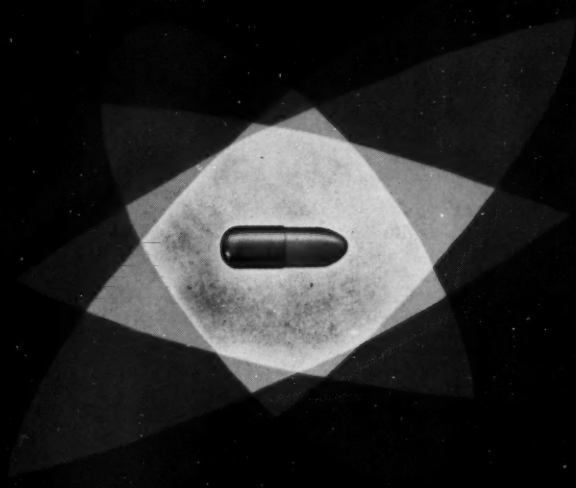
SEBIZON,® antiseborrheic preparation.

Schering

SZ-J-4106

but not a complaint

fastest and shortest-acting oral barbiturate



'Seconal Sodium'

(SECOBARBITAL SODIUM, LILLY)

622011

The secret of sleep in a capsule

When simple insomnia is the presenting complaint, a bedtime dose of 'Seconal Sodium' is often indicated. Its effect is prompt—within fifteen to thirty minutes; relaxation and sleep follow quickly. Your patient awakens refreshed and well rested.

Available in 1/2, 3/4, and 1 1/2-grain pulvules at pharmacies everywhere.

80TH ANNIVERSARY 1876 · 1956 / ELI LILLY AND COMPANY

Darlington Sanitarium

WAWASET ROAD
(Route 842)
WEST CHESTER,
PENNSYLVANIA

A recognized private psychiatric hospital. Complete modern diagnostic and therapeutic services. Qualified physicians may retain supervision of patients.

Moderate rates.

Telephone West Chester 3120

H. VAN DER MEER, M.D., Medical Director MRS. W. J. HANLEY, Superintendent

Borden's
ICE CREAM

After making rounds at
THE DELAWARE HOSPITAL
Stop
and
Visit
Our Newly Remodeled
Store
14th & Washington Sts.
Luncheonette

ACHROMYCIN

ACHROMYCIN*

Tetracycline *Lederle*

for prophylaxis and treatment of

obstetric infections

Posner and his colleagues[1] have reported on the use of tetracycline (ACHROMYCIN) in 96 cases of obstetric complications, including unsterile delivery, premature rupture of the membranes, endometritis, parametritis, and other conditions. They conclude that this antibiotic is ideally suited for these uses.

Other investigators have shown ACHROMYCIN to be equally useful in surgery and gynecology and virtually every other field of medicine. This outstanding antibiotic is effective against a wide variety of infections. It diffuses and penetrates rapidly to provide prompt control of infection. Side effects, if any, are negligible.

Every gram of ACHROMYCIN is made in Lederle's own laboratories and offered *only* under the Lederle label—your assurance of quality. It is available in a *complete* line of dosage forms, including

ACHROMYCIN SF

ACHROMYCIN with STRESS FORMULA VITAMINS. Attacks the infection, bolsters the patient's natural defenses, thereby speeds recovery. Especially useful in severe or prolonged illness. Stress formula as suggested by the National Research Council.

SF Capsules, 250 mg.

SF Oral Suspension, 125 mg. per teaspoonful (5 cc.)

For more rapid and complete absorption. Offered only by Lederle!

dry-filled sealed capsules

[1]Posner, A. C., *et al.;* Further Observations on the Use of Tetracycline Hydrochloride in Prophylaxis and Treatment of Obstetric Infections, *Antibiotics Annual* 1954-55, pp. 594-598.

Lederle

LEDERLE LABORATORIES DIVISION
AMERICAN CYANAMID COMPANY
PEARL RIVER, NEW YORK

*REG. U.S. PAT. OFF.

PHOTO DATA: SPEED GRAPHIC CAMERA, F.16, 1/50 SEC., ROYAL PAN FILM

JOHN G. MERKEL & SONS

Physicians—Hospital—
Laboratory—Invalid Supplies

PHONE 4-8818

801 N. Union Street

Wilmington, Delaware

Physicians' and Surgeons'

PROFESSIONAL Liability Insurance

Provides Complete Malpractice Protection, Avoids Unpleasant Situations By Immediate Thorough Investigation And Saves You The High Costs Of Litigation.

The Only Plan Which Is Officially Sponsored By Your Local Medical Society

The New Castle County Medical Society
The Kent County Medical Society
The Sussex County Medical Society

WRITE OR PHONE

J. A. Montgomery, Inc.

DuPont Bldg. 10th & Orange Sts.

87 Years of Dependable Service

Phone Wilmington 8-6471

If it's insurable we can insure it

ADEQUATE WIRING Certified
REG. U. S. PAT. OFF.

Make Sure You Have Enough "HOUSEPOWER"

"Housepower" is adequate wiring . . . enough electrical power coming into your home to meet the needs of all your electrical uses . . . sufficient number of circuits, outlets and switches for your convenience . . . large enough wires so that the flow of electricity to your appliances is not "choked off."

Be sure your home has adequate "Housepower" for all the appliances you want to use today—and in the future. Adequate wiring for today's modern electrical living . . . serves and saves.

Ask your Electrical Contractor to check your HOUSEPOWER today!

DELAWARE **POWER & LIGHT** COMPANY

DELAWARE STATE MEDICAL JOURNAL

Issued Monthly Under the Supervision of the Publication Committee
Owned and Published by the Medical Society of Delaware

VOLUME 28
NUMBER 11

NOVEMBER, 1956

Per Year, $4.00
Per Copy, $ .50

APLASIA OF RIGHT LIVER LOBE WITH CHOLELITHIASIS

EDWARD M. BOHAN, M.D.*

The patient, a 61 year old white man, was admitted to St. Francis Hospital on October 26, 1955, with the diagnosis of diabetes mellitus. He had two fulgurations for bladder cancer in 1950 and 1954. A transurethral resection was performed in 1954. Physical examination then revealed an enlargement of the left liver lobe, but no further attention was paid to it at that time. Positive serology was noticed in 1950 at the time of his admission for the papillary carcinoma of the bladder.

The diabetes was not a great problem to treat. He did not need insulin. In addition to the diagnosis of diabetes mellitus on the October 26th admission, he had an acute and constant pain in the right chest posteriorly. There was tenderness in the upper right epigastrium, and the initial impression was a pneumonitis of the right base with pleurisy. The main reason for this presumptive diagnosis was the chest x-ray finding: "There is evidence of a localized patch of density in the right lower lobe which gives an appearance suggesting a localized patch of atelectasis, although this may represent an area of pneumonitis. The changes over the base are consistent with a slight pleural effusion or localized pleural thickening". (Peter J. Olivere, M.D., Radiologist)

Therefore, the original impression of cholecystitis was somewhat shaken. Icterus index was 11 units, and cephalin flocculation was 3+ after 48 hours. Flat plate of the abdomen revealed no pathology. Cholecystogram showed a poorly or nonfunctioning gall bladder: no evidence of any radiopaque calculi.

Another x-ray examination of the chest was made on 11/2/55. A clearing of the changes at the right base was not reported. Electrocardiogram was normal. Kahn test was 2+.

A gastrointestinal x-ray study showed nothing of note except pylorospasm and perhaps reflex disturbance in motility of the upper gastro-intestinal tract. The colon also appeared within normal limits on barium enema x-ray.

Intravenous urogram showed good function in both kidneys with a mild degree of ptosis of the left kidney in the erect position. There was no evidence of calculi. This film was taken because of the patient's past bladder history, and the possibility of a hydronephrosis on the right side.

DIAGNOSIS FINALLY MADE

On December 27, 1955, this patient was readmitted to St. Francis Hospital with the diagnosis of cholecystitis with jaundice and diabetes mellitus. He had abdominal pain, nausea, and vomiting. He was subject to belching after ingestion of food. He had one white stool, and his urine had been very yellow. His blood sugar, two hours postprandial, was 192 mgm./100 cc. of blood. CO_2 was 47 volumes %, and prothrombin time was 100%. A direct van den Bergh test was 0.9 mgm., indirect van den Bergh 3.5 mgm. Total bilirubin amounted to 4.4 mgm., and icterus index was 40 units. Serum amylase amounted to 80 units and alkaline phosphotase, 13.1 Bodansky units.

A complete examination of cerebrospinal fluid was made. The colloidal gold reading was 1-1-1-1-2-1-0-0-0-0. Protein was 42 mgm., chloride, 700 mgm., and the Wassermann was weakly positive. Culture of the spinal fluid was made after 24 hours. Urine urobilinogen was positive in dilution 1:40.

* Chief of Medicine and Metabolism, St. Francis Hospital.

This report is about a diabetic patient who developed pain in the chest and upper right side of the abdomen. Symptoms, signs, laboratory and x-ray findings were at first confusing. Diagnosis was partially made before operation, but an unsuspected absence of the right lobe of the liver forced the surgeon to do a cholecystolithectomy in the chest. It was impossible to remove the gall bladder. Review of the literature (1939-1956) revealed no similar case. Due to the agenesis of the right lobe of the liver, this patient's symptoms and signs were very unusual. A problem in differential diagnosis was presented which would be rarely encountered in the practice of medicine. It was not until the jaundice manifested itself that we were sure of our preoperative diagnosis.

Urine bilirubin was negative, and urine urobilinogen total for 24 hours was 2.9 mgm. Urobilinogen in the feces was slightly positive.

The diabetic state was now somewhat disturbed. The blood sugar ran up into the 300 mgm. level. There was no disturbance of CO_2 at this time.

A chest consultant felt there was no chest disease, despite the original x-ray findings.

A neurologic consultation was reported as follows: "No evidence of any intraspinal lesion can be found as a cause of this patient's pain".

Since the consensus of opinion was chronic cholecystitis, probably with stones, decision was made to operate. The following is the operative report:

Findings: The gall bladder was represented by a firm, thick, fibrous, shrunken sac containing a solitary stone about the size of a pullet's egg. The stone filled the entire lumen of the gall bladder, and apparently was the cause of obstruction which prevented the gall bladder dye from entering the viscus at the time of cholecystography.

There was apparently a congenital absence of the right lobe of the liver. The left lobe of the liver was somewhat larger than average normal size, and extended across the epigastrium, having a somewhat rounded edge and a nutmeg appearance, suggesting some chronic congestion. Due to the absence of the right lobe of the liver and the very high position of the gall bladder, difficulty was encountered in exploring this area. Due to the congenital anomalous conditions, it was not possible to state with certainty whether the common duct was patent or not, and no attempt was made to explore the duct because of its inaccessibility at a very high level. It was hoped that drainage of the gall bladder by cholecystostomy tube would afford relief of the biliary tract infection.

Procedure: A transverse incision was made at the level of the 9th costal cartilage across the upper right abdomen. Abdominal layers were opened transversely; vessels were clamped and tied with #35 steel. Upon opening the abdomen, the congenital absence of the right lobe of the liver was first discovered, and the gall bladder was found to be located beneath the dome of the diaphragm at the level of the 8th rib.

Incision was then enlarged upward and laterally, extending between the 10th and 11th ribs, and a rib-spreader retractor placed to afford exposure of the gall bladder area. Dense fibrous adhesions were separated by division between clamps and then ligated with #32 steel.

The gall bladder was opened, and a large soft stone removed with stone forceps. The gall bladder was mopped out with gauze, and a #20 mushroomcatheter placed in it and held in place with a pursestring suture to close the fundus around the tube. Gelfoam was placed in the region of the gall bladder to control oozing from the liver surface, and a large soft rubber tube drain placed beneath the diaphragm on the right side, near the gall bladder. Both the cholecystostomy tube and the other rubber tube drain were brought out by the lateral angle of the incision between the 10th and 11th ribs, and anchored to the skin with a suture of #32 steel. The ribs and abdominal wall were closed in layers around the two drains with #32 and #35 steel; dry dressings applied.*

The patient's post-operative course was quite rough, but not from a diabetic standpoint. He was kept on ginger ale, orange juice, clear broth, and black coffee, and given two intravenouses daily of distilled water with 10% Travert, plus 20 units of plain insulin. He became somewhat psychotic and mentally removed from his environment. However, on January 9th, he was placed on a semi-soft diabetic diet, and Novogran was added to the intravenous therapy. His post-operative course from January 18th was much better. He slowly recovered mentally, and he was able to get into a wheel chair on this date. No evidence of an electrolytic disturbance was found on repeated examination.

REVIEW OF THE LITERATURE

Complete absence of the left lobe of the liver is extremely rare. Reddy[1] reports a case found at autopsy with arrest in the formation and development of the left embryonic bud of the liver as early as the 3 mm. stage.

* Surgery performed by Dr. Daniel J. Preston, Chief of Surgery, St. Francis Hospital.

Cullen[2] reports that it is unusual to find the right lobe reduced in dimensions and volume. With absence of the diaphragm, the right lobe may be transposed to the left side or the right lobe may be in the thorax. No case similar to this can be found in the medical literature (1939-1956).

SUMMARY

Looking at this case in retrospect makes the diagnosis very easy. Let us consider the impression of December 28, 1955. In a note made to the surgeon, Dr. Daniel Preston, I stated "This 61 year old diabetic of ten years has had severe pain in the lower right chest and upper abdominal region for about two months. This is his fourth admission to this hospital. On each admission, he recovers from this pain, but it recurs a week later after discharge, and becomes increasingly severe. At no time has there been a history of cold or cough.

"My first impression was gall bladder pathology, but when the x-ray of the chest was reported, the diagnosis seemed more likely to be virus pneumonia with pleurisy. However, the persistent recurrence of the symptoms suggested that we were dealing with an odd condition.

"We would like to rule out any abdominal pathology. Of course, diabetic neuropathy is also a possibility, but the symptoms seem too severe for this. In addition to the possibility of gall bladder disease, the possibility of liver pathology has been entertained, or diaphragmatic hernia. No jaundice was present on previous admissions, but jaundice is now appearing.

"By a process of elimination, the chest was ruled out as a cause of the patient's pain."

The positive serology made the case still more confusing, and even the possibility of girdle pains was thought of. By means of the liver profile, the next note was made by the author on January 1, 1956:

"The elevation of the blood cholesterol and alkaline phosphatase points strongly to obstructive jaundice. Patients with bile duct obstruction due to carcinoma have higher total cholesterol values usually than when the obstruction is due to calculus. The pain is also suggestive of stones. We have been giving this patient a complete course of penicillin, so that the positive serology might be taken care of."

On January 4, a new note was made as follows: "Urobilinogen is not increased in urine. This further points to obstructive extra-hepatic jaundice. I believe we can now go ahead for surgery."

We could never ascertain why the patient became temporarily mentally deranged. His post-operative blood urea was slightly elevated, and the icterus index was still 30 units. Undoubtedly, he had some liver decompensation.

Since the history of a positive serology went back many years, it is believed that this disease was in a quiescent stage. However, this is pure speculation.

The patient made a slow but spectacular recovery at home, and by the first of July was ready to return to work. The liver is still enlarged on the left side. This may have always been the case, perhaps since birth.

REFERENCES

1. Reddy, D. J.: Complete absence of left lobe of liver, Journal of the Indian Medical Association, 23:9, 405.
2. Cullen, T. J.: Accessory lobes of the liver, Arch. Surgery 11:718, 1925.

THE PRESENT STATUS OF THE TREATMENT OF RHEUMATIC DISEASES*

PHILIP R. TROMMER, M.D.**

In this presentation the rheumatic and para-rheumatic disease other than rheumatic fever will be discussed. Included in this group are the so-called collagen diseases, gout, bursitis, degenerative (osteoarthritis), sub-luxation of the joints and the shoulder-hand syndrome.

The so-called collagen diseases derive their terminology from the work first done by Klinge and then called such by Klemperer and Baehr. The authors today probably feel that the term is not sufficiently inclusive since collagen is but one part of the system, and the label of mesenchymal tissue disease (embryologic) or connective tissue disease is more appropriate. The diseases included in this entity are rheumatic fever, rheumatoid arthritis, lupus erythematosus, dermatomyositis, scleroderma and

* Presented at Scientific Meeting, St. Francis Hospital, March 27, 1956.

** Assistant Clinical Professor of Medicine, Woman's Medical College of Pennsylvania; Associate in Medicine, Albert Einstein Medical Center, Southern Division, Philadelphia.

polyarteritis nodosa. Although these disease may have arthritis as a common presenting symptom, the disease entity may not show its true nature for as long as three months to years. Therefore the term undifferentiated mesenchymal-connective-tissue-collagen tissue disease has been introduced. This term implies a widespread systemic involvement of the connective tissue not only in the joints but also in the skin, muscle and visceral organs of the heart, lung, spleen and intestinal tract and even the eyes. A similar clinical picture may often be the presenting symptom complex in a malignant process.

RHEUMATOID ARTHRITIS: This disease is included in the "collagen tissue diseases" and involves connective tissue other than just those in the joints. As such, when the spleen is involved with the presence of a neutropenia it is called Felty's Syndrome. The unfavorable process may also involve the eyes in the form of iritis. In fact, the latter may be the presenting pathology. Involvement of the eyes resembling a malignant tumor has been reported in which enucleation was had because the true pathology was not recognized.

For an earlier and more specific diagnosis, the sensitized sheep red cell test is being evaluated.

The principles of treatment are bed rest, preferably in a hospital, with graded and graduated physio-therapy aimed towards the eventual rehabilitation of the patient and the prevention of deformities. This requires the close cooperation of the services of the medical, orthopedic and physical medicine branches of medicine. Transfusions of 250cc of whole blood, plasma or 48 hour post-partum plasma twice a week is most helpful in correcting the anemia, supplying protein and "antibodies".

SALICYLATES in the form of acetyl-salicylic acid or its equivalent in doses graduated up to 6-9 grams per day is incated as the drug of choice. This should be exploited for 3-6 weeks to determine its efficacy. A recent survey by the British Empire Council of Rheumatic Diseases has shown that aspirin and steroids appear to be equally effective in at least 50% of the cases and to give the same results after two years. The second drug of choice added to salicylates and which will produce a remission of 50-80% of cases is a gold salt.

The preparations used are gold sodium thiosulphate (Searle), Solganol B (Auro-Thio-Glucose) (Schering), Myochrysine (Auro-Thio Malate) (Merck) and Lauron (Auro-Thio-Glycoanilide) (Endo). Today all are administered intramuscularly. Various dosage schedules have been advised. The author has been following and advises a dosage of only 10 mg. per week. With such a small dosage, the adverse effects of the liver, kidney, skin and hematopoietic system, except for sensitivity, are avoided. Red, white and platelet counts, together with a microscopic urinalysis, are done weekly for four weeks; biweekly for four weeks, and thereafter once a month as long as the patient is being given a gold salt, British anti-lewisite (BAL) is the antidote for gold toxicity.

Physio-therapy in the form of hydrotherapy, Kenny or hydro collator hot packs, removable splints, graded and graduated exercises are carried out daily until the patient is ambulatory and thereafter as needed until fully rehabilitated.

Proper hygiene, diet and vitamins are included in this program.

The last group of drugs one thinks of using are those of the steroid group. These drugs include ACTH, Cortisone, Hydrocortisone, Prednisolone and Prednisone and another with a similar action—Phenylbutazone. The steroids have the superior advantage of quick action-relief of pain, subsidence of inflammation, early ambulation, reduction of the sedimentation rate, but the disadvantage of the prompt recurrence of symptoms after cessation of use. The side effects of these drugs must be considered, since they produce a retention of sodium, a loss of potassium and calcium, and an increase in the blood volume with a rise in the blood pressure, hyperglycemia and thrombophlebitis. Exclusive of ACTH and Phenylbutazone, these drugs place the patient in the dangerous position of a hypoadrenal status akin to an Addisonian Crisis with the patient most vulnerable to severe shock states on the slightest provocation of

stress in the form of infection (which they help to spread) and injury.

Since ACTH has to be given intramuscularly its disadvantage is obvious. The oral dosage of the steroids varies with Cortisone up to 35-50 mg. per day, Hydrocortisone, 15-25 mg. per day, Prednisolone and Prednisone 2.5 to 15 mg. per day and Phenyl-Butazone 100-300 mg. per day. The steroids are contraindicated in infectious states (unless covered by a proper antibiotic), ulcer cases, diabetes (requires extra insulin) and in hypertension and cardiac decompensation (may be used with mercurials).

Intra-articular hydrocortisone acetate daily or weekly or the tertiary butyl acetate in dosage of 5-50 mg. (dependent upon the size of the joint) may be added to the salicylate-gold salt regime. This has the distinct advantage of being locally effective in reducing pain, inflammation, joint swelling and aiding rehabilitation without the disadvantages incident to the systemic use of the steroids.

In certain cases codeine may have to be used since pain is one of the most disabling features of the diseases.

RHEUMATOID SPONDYLITIS: The central form of rheumatoid arthritis is thought to be a different disease from peripheral rheumatoid arthritis because of its incidence (9 males to 1 female), nonresponse to gold salts, muscular pain relief by x-ray therapy and the negative reaction of the sensitized sheep red cell test. The most efficacious form of therapy previously utilized was x-ray and physio-therapy. The preferred choice will most likely be that of the use of steroids and physical therapy. Reports from abroad have shown an increasing evidence of leukemia late in life attributed to the x-ray therapy. This also leads to a reduction in the patient's x-ray tolerance "bank". In children this disease appears to run a self-limited course from six months to three years so that steroids and not x-ray are best indicated, along with the time-honored physiotherapy procedures.

GOUT: is still considered to be a disease of altered metabolism. Recent radioactive studies have isolated the source of each of the carbon and nitrogen atoms in the molecule. As such, uric acid is now known to be formed from carbon dioxide, formate, lactate, glycerine, serine and threonine. Also the so-called miscible pool of uric acid is present in the body and totals from 700-1300 mg. with a daily turnover rate of 50-75% in the normal individual and a reduced rate in the patient with a gouty diathesis.

The rule of thumb is to consider that every attack of ACUTE MONOARTICULAR ARTHRITIS IS GOUT UNTIL PROVEN OTHERWISE. As such, every case of a monoarticular arthritis following surgery or injections is also gout until proven not to be so. If this rule of thumb is followed a "heroic and erudite" diagnosis will be made and relief quickly provided to a patient who will be most grateful.

TREATMENT OF ACUTE GOUT: The time-honored therapeutic test and treatment of the acute attack is the proper use of colchicine alkaloid .65 mg. (gr 1/100) every hour on the hour for 10-15 doses or even less when relief is obtained. Nausea, vomiting and diarrhea ensue. Thereafter, 4 cc. of paregoric is given for each bowel movement. For some unknown reason toxicity and therapeutic efficacy are closely allied. No other form of acute arthritis responds to colchicine therapy. When the number of doses are known to produce relief the subsequent attacks can be treated with a reduction of one or two doses and toxicity may thereby be reduced. Relief may be expected in 24-72 hours. Recently a new form of colchicine that of des-acetyl methyl colchicine (1 mg.) given orally from 1-4 doses every 5-8 hours has been reported to be efficacious without causing nausea, vomiting or diarrhea.

Intravenous colchicine administered in a single dose of .25 to 3 mg. in normal saline has been reported to be safe, rapid and effective without producing gastro intestinal toxicity.

PHENYLBUTAZONE (BUTAZOLDIN) (R) will most likely replace colchicine as a therapeutic test since it does not produce the toxic side effects and is a quick saturating one of 4 tablets of 100 mg. each and followed by a 100 mg. tablet every three hours until relief is had. Since this is a short term form of therapy, the feared

hematopoietic blood complications are not so great. ACTH 40-80 units (either plain or in the gel form) will give relief in as quickly as two hours. However it is followed by a rebound phenomena and must be supplemented with at least 3-4 doses of colchicine per day for three to four days.

Local relief may also be obtained by the immobilization of the involved joint and the application of COLD and not heat as is so often done. Ice compresses will often obviate the use of a narcotic for relief of pain. The generally recognized diet is to restrict food to cottage cheese, skimmed milk (fats cause a retention of uric acid), carbohydrates and plenty of fluids.

(2) RECURRENT - CHRONIC - INTERVAL GOUT is treated by the dietary restriction of nucleoporteins, deprivation of any food that the patient may be "allegric" to or is known to cause an exacerbation, and the free use of fluids to prevent uric acid kidney stones. The hyperuricemia may be reduced by a restricted nucleoprotein diet, salicylates up to 6 grams per day or Probenecid (Benemid (R)) from .5 grams to 1.5 grams per day. The efficacious action of Probenecid on the urinary tubules in preventing the reabsorption of uric acid is impaired by salicylates. Recurrent attacks can also be prevented by using colchicine .65 grams three times a day.

(3) CHRONIC GOUTY ARTHRITIS and uric acid kidney stones are best treated by the free use of fluids and Probenecid .5 grams per day. Any higher dosage of Probenecid may mobilize too much uric acid and cause an attack of gouty arthritis or kidney stones. It is best to "peel off" a bit or layer of uric acid slowly, over a six months to one year period, than to have a quick mobilization. Acute attacks and kidney stone complications are thus prevented. Every patient with kidney stones requires a chemical analysis of the stones and if they are of uric acid composition, gout should immediately be suspected and treated accoringly. Surgery may be necessary for the removal of large annoying deposits of tophi.

A patient with gout should always carry colchicine or Phenylbutazone with him just as he does his keys. At the first sign—"twinge or twist" of joint involvement he should proceed just as in the treatment of an acute attack and use the drugs to "abort" a full-blown seizure. With relief, therapy can be stopped without having to go through the full schedule.

All cases of cholelithiasis and nephrolithiasis should have the stones analyzed for uric acid composition, hyperuricemia and gout.

BURSITIS, PERI-ARTHRITIS (SHOULDER-CUFF) TENDONITIS & TENDO-VAGINITIS: Not all cases of so-called bursitis are really so. Very often the involvement is not in the bursae, but may be a tendonitis, or a vagino-tendonitis and still present the clinical picture of a frozen shoulder. Calcification does not always occur. The newly recognized treatment of efficacy today is to treat the condition as a localized sterile abscess with the application of cold and rest for 48-72 hours and heat and mobilization thereafter. Daily local hydrocortisone acetate 25-50 mg. injected along with Procaine in the manner of an inverted cone in the area of localization (not necessarily in the bursae) will often result in relief of pain, (irritative exacerbation may occur) increase in motion range, absorption of the calcific deposit and avoidance of the frozen shoulder. My-B-Den (Bischoff) has been reported to be helpful. The other steroids and phenylbutazone are also used, along with codeine and salicylates for relief of pain and restoration of function. X-ray therapy has been advocated by the roentgenologists, whereas many medical men are not convinced of its efficacy. However, in a severe case the patient deserves to be given all known "forms of therapy" (have the book thrown at him). It is not very well recognized that a "frozen shoulder" can occur with or without calcification and that the residual muscle spasm requires vigorous and intensified physiotherapy for as long as necessary to produce a normally functioning shoulder. Surgery for the removal of the calcific deposit and mobilization of the shoulder cuff may be necessary in a small percentage of cases.

REFLEX SYMPATHETIC DYSTROPHY OR THE SHOULDER HAND SYNDROME: The latter name was given to a group of cases by Dr. Steinbrocker with involvement of the hand and shoulder resembling unilateral arthritis and a frozen shoulder with eventual disability in many cases. In many cases it follows myocardial infarction and visceral involvement of the chest. The syndrome has also often been confused with a recent coronary occlusion and myocardial infarction. It may be present without any known etiology. Treatment is directed to the underlying etiology and followed by that advocated for bursitis, plus sympathetic nerve block and sympathectomy. This condition is often most painfully disabling mechanically and may even resemble causalgia.

SUB-LUXATION of the CERVICAL and SACRO-ILIAC JOINTS: Following a "whiplash" effect most commonly produced by even the slightest rear-end auto collision the neck is snapped back and forth. This often results years later in a degenerated cervical disc or even subluxation. The latter, when it also involves the sacro-iliac joints can be relieved by a qualified, erudite practitioner who understands the principles and limitations of manipulative therapy. There is a great deal of controversy by anatomists and orthopedists as to whether such a condition can occur or even exists. The author has seen and successfully treated too many of these cases by manip ulation so that the discussion is really academic. There is no need for these patients to be treated by medically unqualified cultists wherein a proper diagnosis is most necessary. Neither is there any need for the eyes and ears of the medical profession to be closed and covered to the existence of such a condition. Needless to say careful physical and roentgen examination is a prerequisite to such a method of treatment.

HEMOLYTIC REACTIONS IN BLOOD

MARIO LEON, M.D.*

Causes of hemolysis in blood transfusion reactions are extravascular and intravascular.

* Resident Physician, St. Francis Hospital.

EXTRAVASCULAR REACTIONS: These are caused by improper handling of the donor's blood before injection. Freezing or overheating may cause hemolysis. The blood should be kept between 4° and 6° C. in a refrigerator. Improper storage may occur by keeping the blood at room temperature or for too long a period of time at the above specified temperature. The blood should be immediately chilled and constantly stored at the low temperature until used. With increasing storage, the erythrocytes become increasingly fragile with a tendency to more severe reactions of the hemolytic type. Stored blood is believed inferior to fresh blood for controlling hemorrhage in jaundiced individuals.

INTRAVASCULAR CAUSES: Stored erythrocytes carrying the Rh factor may produce reactions in individuals who have had previous transfusions. Multiple transfusions with Rh positive corpuscles may produce an agglutinin. The first and subsequent of women in pregnancy and the puerperim must likewise have Rh negative corpuscles.

The majority of intravascular hemolytic reactions are not serious, usually being characterized by fever and chills and possibly hemoglobinuria and jaundice; but severe reactions due to gross incompatability of the bloods of the donor and recipient are serious and commonly are known as *hemolytic shock reactions.* However, they are not necessarily fatal, even when large amounts of blood are transfused. The symptoms include violent pains in the back, chills, respiratory embarassment, circulatory collapse, hematuria, hemoglobinuria, jaundice, urticaria, and symptoms due to small hemorrhages or emboli in the brain, mesentery, endocardium and the gastrointestinal mucosa. The terminal stages of those who survive the immediate effects are characterized by renal failure. Probably 1 to 5 hemolytic reactions per 1,000 transfusions are caused by human error.

Undoubtedly, there is far more danger of serious reactions by transfusing corpuscles incompatible with the plasma of the recipinet than transfusing plasma incompatible with the corpuscles of the recipient. The latter may result in intravascular agglutina-

tion and hemolysis. It is important to differentiate severe reactions from non-specific causes from those due to incompatibility. Repetition of the grouping and cross-matching tests on the blood taken before transfusion and also on fresh blood may determine whether there has been an error in grouping. The blood taken from a patient after transfusion could also be examined for hemoglobinemia and icterus index; the urine should also be examined.

RENAL INSUFFICIENCY FOLLOWING BLOOD TRANSFUSION REACTION: Many recipients who develop hemoglobinemia continue to secrete urine satisfactorily, and appear well within 24 hours after transfusion. Transient albuminuria usually accompanies the hemoglobinuria. The volume of urine may be increased during the stage of hemoglobinuria.

The early symptoms are followed by progressive lassitude, prostration, nausea and vomiting. Frequently the patient becomes irrational. Convulsions may occur. At times, coma and death occur between the 4th and 15th day after transfusion. Daily urine volume is between 800 ml. and 0.

Inexperienced clinicians are wrong in thinking that a urinary output of 500 ml. per day excludes the possibility of renal insufficiency. The arterial blood pressure usually remains normal, although a few cases are hypertensive. During the period of oliguria and anuria, diuresis may occur and recovery follow. This is accompanied by the gradual disappearance of the azotemia. On each day of the renal insufficiency, the blood urea and creatinin increase, although the greatest increase is noted during the first 24 hours.

TREATMENT OF THE PRIMARY SHOCK: The renal ischemia accompanying the hypertension of the primary shock might predispose to tubular damage from hemoglobin, and measures should be taken to raise the arterial blood pressure. Subcutaneous injections of either Epinephrine Hydrochloride or Ephedrine Sulfate will help maintain normal levels of blood pressure. Nor-Epinephrine has been a valuable adjunct to this group of medicines, and is given intravenously.

The renal anuria is difficult to treat, but one may proceed by discontinuing the transfusion. Intravenous fluids should be used to replace those lost by vomiting and sweating and to correct any electrolyte disturbance brought about by the shock. Promotion of circulation through the kidney may be helped by this fluid replacement, diathermy and irradiation of the kidney region, transfusion of a compatible blood, and, in rare cases, decapsulization of the kidneys. Spinal anesthesia has been used with very uncertain results. Some doctors believe in alkalinization of the urine. Peritoneal irrigation has been used.

One must remember that circulatory overload accounts for more deaths than hemolysis. The best treatment is prophylaxis.

PREVENTION OF REACTIONS to blood:

1. Care in the storage and handling of donor's blood.
2. Meticulous attention to labeling of flasks and keeping of records.
3. Competent compatibility tests before transfusion.
4. Prompt termination of transfusion when symptoms of complication occur.
5. Thorough investigation of causes of hemolysis.
6. Treatment of recipient in the phase of primary shock.
7. Alkalinization of the urine as soon as hemolysis is suspected.

ASPECTS OF ALCOHOL INTOXICATION

Compiled by

FREDERICK KNECHT, JR., LL.B.

INTRODUCTION by H. Thomas McGuire, M.D., Director, Department of Medicine, St. Francis Hospital:

The emergence of alcoholism to its proper consideration as a portion of a disease entity has reawakened our medical responsibility in this very serious and challenging problem.

Alcoholism's impact upon society has necessitated reassessment of all areas of responsibility: community, as well as medical and legal. The author of the following paper has shown interest, perceptiveness, and thoroughness in his presentation of alcoholism and law. We are indebted to him for this contribution.

ALCOHOL AND THE CRIMINAL LAW*

It sometimes happens that a man who is under the influence of drink (or other drugs) will commit crimes that he would not have thought of committing while so-

* Presented at Medical Conference, St. Francis Hospital, October 4, 1956.

ber. This is not because of the supposed stimulating effect of alcohol, for it seems that the action of alcohol on the cerebral cortex is depressant from the beginning. What alcohol does is to remove inhibition or self-restraint, and impair the appreciation of the consequences of conduct. It may also conduce to crimes of negligence by impairing powers of perception, delaying reaction-time, and rendering movement clumsy.

The law on the subject of drunkenness as an excuse for crime is simply this: that there is, in general, no special law on the subject of drunkenness. The rules are for the most part applications of ordinary principles of the criminal law. Merely to show that a man's mind was so affected by drink that he more readily gave way to a violent passion is no defense. Similarly, it seems that a drunkard receives no special consideration in assessing negligence. Although drunkenness amounts to insanity on some definitions of that word, the two are legally distinguished. Insanity in law means disease of the mind other than aberration of a temporary nature produced by drugs. Drugs, though they may bring about a dual personality, are largely ignored by the law, so that, it is said, Mr. X sober must pay the penalty for the misdeeds of Mr. X drunk.[1]

To the general rule there are two exceptions, and these are the only two instances where any special exemption has been recognized for drunkards:

Habitual drinking may occasionally lead to such permanent change in the brain tissues as to be accounted insanity, such as *delerium tremens* and alcoholic dementia. That the case is not in analysis one of mere drunkenness is shown by the fact that an attack of *delerium tremens* may occur at a time of complete abstinence. Conversely, alcohol addiction is recognized by experts as in some cases a symtom of mental illness; and such disease may be latent, brought out by the consumption of alcohol. For instance, alcohol may excite an epileptic seizure.

If the defense succeeds before a jury, the accused must be sent to a mental institution. Therefore, this defnse is not frequently used in practice because the accused or his advisor is apt to fear that the detention in a mental institution will be longer than an ordinary term of imprisonment for the crime. However, the Canadian view is that if a defense of drunkenness is set up, the judge may direct the jury on insanity notwithstanding a denial of insanity by defending counsel.[2]

It has been held that even temporary insanity produced by drink has the same effect as permanent insanity.[3] No indication is given, however, of the difference between temporary insanity and drunkenness.[4] Assuming a verdict of "guilty of the act but insane", the form of treatment presents difficulty, if the accused is perfectly sane when not under the influence of drink. Incarceration in an ordinary mental institution would hardly be proper. It might be suitable to release some offenders on probation with a condition to refrain from alcohol; yet probation is impossible on a verdict of insanity. In other cases institutional treatment may be indicated; but there are no disciplinary institutions for alcoholics. The problem of justice is well illustrated by the case where a man was convicted of murdering his wife during a drunken debauch. He reacted morbidly to alcohol, having an insane heredity aggravated by sunstroke; and the killing had left no trace on his memory. He was sentenced to penal servitude. There was no ground for doubting the genuineness of the amnesia, which persisted throughout the period of his penal servitude, when no motive for simulation any longer existed.[5]

Involuntary drunkenness is a defense, as where the accused's companions put alcohol into his ginger-beer,[6] or where he is unwittingly incapacitated by medicine.[7] Baron Parke in the case concerning the ginger-beer remarked: "If a party be made drunk by stratagem, or the fraud of another, he is not responsible." The courts, however, interpret "coercion" in this connection quite literally to mean that one was "forced . . . to drink" [8] by overt physical acts. "Fraud" which is generally equated with force in the criminal law, is also so rigorously restricted

as to exclude, for example, imposition upon the young and inexperienced by calculating adults.[9] In Perryman v. State, 12 Okla. Cr. App. 500, 159 Pac. 937, it was said: "Involuntary intoxication is a very rare thing and can never exist where the person intoxicated knows what he is drinking and drinks the intoxicant voluntarily, and without being made to do so by force or coercion." Also in Burrows v. State, 38 Ariz. 99, 116, 297 Pac. 1029, 1036 (1939), the court held that involuntary intoxication "must be included by acts amounting in effect to duress." Finally, although it has been stated that "taking liquor prescribed by a physician" is a defense,[10] a court was unwilling to entertain the plea that the defendant drank to obtain relief from an acute pain.[11]

From these cases it is appearant that Baron Parke's statement above has been narrowly limited by the American courts. This seems unfortunate because a broader interpretation may be justified for the reason that the plea of involuntary drunkenness is not open to the same abuse as one of voluntary drunkenness, and the offense is not so likely to be repeated. The amazing thing about the factual situations met in the decisions on intoxication, which, because of the usual judicial stress on fraud and coercion as exculpatory becomes appearant only after close study of the cases, is that involuntary intoxication is simply and completely nonexistent. The reports record hardly a single decision actually holding that the defendant was involuntarily intoxicated, and the opinions invariably hold that the intoxication was "voluntary". However, with equal uniformity they reiterate that fraud or coercion is a defense. "Fraud" narrowly interpreted in the cases to require complete innocence of the nature of alcoholic drink, wrongfully induced, cannot even be perpetrated on normal children at the age of legal capacity. As regards "coercion", the case-law implies that a person would need to be bound hand and foot and the liquor literally poured down his throat, or that he would have to be threatened with immediate serious injury before the exception, so universally voiced, would have an effect on judicial decision. So far as the present law is concerned, there would be no difference in the actual rule if it were announced flatly that no intoxicated person could ever claim that his condition was effected by fraud or coercion. However, the principles, signifying that voluntary conduct may be punishable but that involuntary conduct is not within the reach of punitive sanctions, are not impeached because judges continue the routine of invoking the precept that "voluntary drunkenness is no excuse" in circumstances where it is plain that the drunkenness was anything but voluntary in any realistic connotation. This serves to transform the avowed "exceptions" into mere dicta which functions only as a balm for the judicial conscience, rationalizing the infliction of severe penalties.[12]

Nor is the validity of the fundamental principles impaired because judges, untouched by modern science in these matters, hold many persons liable, though they are in fact afflicted with mental disease. A rather frequent defense is that a serious injury predisposed the defendant to alcoholic addiction. It is claimed that a fractured skull,[13] a blow on the head,[14] an injury to the brain,[15] or some other serious accident[16] stimulated indulgence or resulted in complete lack of control. These claims are uniformly rejected by the courts, and the defendants are treated as normal. However, it has been reported that persons "having severe head injuries and sunstrokes, are particularly predisposed" to mental disease when under the influence of alcohol.[17] Yet the courts insist that this is in law no excuse whatever. On the contrary, such a person is held even more culpable because, so it is argued, knowing his weakness, he should have put forth greater efforts to obstain, and convictions, even of murder, are upheld in such cases. Although this judicial logic is superb, the premises are fallacious because they are formulated without regard to available medical knowledge, for in these cases the claim of a serious head injury is typically accompanied by well-marked pyschopathic symptoms of incapacity during sobriety as well as by sharp deterioration under intoxication. Nevertheless, even if the defendant

is believed to have been temporarily insane, he is held punishable because it is an artificial voluntarily contracted madness. It was said in Choice v. State, 31 Ga. 424, 480-481 (1860): "A man may have partial or general insanity, and that, too, from blows on the head, yet if he drink, and bring on temporary fits of drunkenness and while under the influence of spirits, takes life, he is responsible."

In many cases it is clear that the defendant is mentally diseased. These are usually homicides where the motive for killing was obviously irrational. Yet in spite of this evidence being introduced, the courts continue to hold that temporary insanity, occasioned immediately by drunkenness, does not destroy responsibility for crime, where the defendant, when sane and responsible, voluntarily made himself drunk. The theory is that unless the defendant is "permanently" insane, his mental condition should be entirely ignored on the inhibiting assumption that the intoxication was "voluntary". As was held by a Michigan court: "He must be held to have intended this extraordinary derangement . . . and the other results produced by it".[18] Such decisions are not rare, but rather they represent the settled judicial attitude. However, in most cases of this nature the clinical picture bears unmistakable marks of mental abnormality.

Thus we find competent courts ignoring serious physical injuries, addiction, chronic alcoholism, *delerium tremens*, any psychoses because accompanied by intoxication and, in general, adhering to a course of adjudication that can only be regarded as unenlightened. The combined result of narrowing "fraud" and "coercion" to the vanishing point when dealing with normal persons and refusing to acknowledge that many defendants are diseased, implemented by tradition, and, in its proper place praiseworthy, disapproval of drunkards is harsh law that adds cruelty to misfortune. The personal remorse consequent on realization of what happened is exceeded by the social tragedy of severe punishment for harms committed under severe intoxication, not infrequently by persons who are seriously diseased. Yet it is clear that able judges have tried to alleviate the rigor of the criminal responsibility of inebriates. It is equally plain that the continued unsatisfactory state of the law is in large part due to the methods employed to achieve the desired mitigation. Confusion is the resulting product from the application of the exculpatory doctrine as well as formalism and rigor of interpretation of "voluntary intoxication".

As a matter of history the true reason why drunkenness was disregarded as a defense was because it was thought that drunkenness being a voluntary act, and being, moreover, immoral if not illegal ought not to be an excuse. It was on this principle that involuntary drunkenness was regarded as excusing. Yet insanity, which excuses from punishment, may sometimes be quasi-voluntary. It is well known that syphilis may result in insanity (general paresis), and the contraction of syphilis may be the result of a conscious running of risk; but the sufferer from general paresis receives the same exemption as any other insane person. However, the casual connection between sexual intercourse with an infected person and general paresis is more prolonged than that between drinking and drunkenness.

Another aspect of alcohol intoxication in the criminal law is that even if the accused was drunk at the time of the crime, it must be shown that he performed the act and that he had the mental state requisite for the crime. If the crime requires intention, drunkenness may help to show that there was no desire of the consequence; if it requires recklessness, drunkenness may help to show that there was no foresight; drunkenness may also help to negative knowledgs or establish mistake. In short, drunkenness is a matter to be taken into consideration in deducing intent or recklessness from outward acts. There is no special rule here, except that drunkenness is admissible in evidence to show that the mind was eclipsed. Suppose that a man when drunk commits some crime that (1) he would not have done when sober, (2) he did not desire to do when he became drunk, and (3)

he did not know himself to be capable of doing when drunk. Under the law as it stands today, proof of these facts would be admissible in mitigation of punishment.

In accordance with the same principle, drunkenness has been admitted as a factor in evidence to show that a man did not intend to kill,[19] to commit suicide,[20] to do grievous bodily harm,[21] or to attempt a crime.[22] But in the nature of things, drunkenness cannot normally negative an intent to rape, for rape cannot normally be committed unintentionally; consequently, a killing in the course of rape will in law be murder. Drunkenness could be relevant in rape only if it resulted in a mistake of fact, as if it made the accused think that the woman was consenting or was his wife. These propositions, true for alcoholic intoxication, are, of course, just as applicable to other drugs.

The law is sometimes stated in a restrictive form, it being said that drunkenness may help to negative a "specific intent". Lawyers tend to breathe this phrase with peculiar reverence, but it is suggested that the word "specific" is inaccurate. If the word means an intent expressly alleged in the indictment, the rule is too narrowly framed, for no intent is specifically alleged in an indictment for murder, yet an intent is involved and drunkenness may help to negative it. It makes no substantive difference whether an intent is required to be expressed in the indictment or is implied in the name of the crime. All that is required for the present purpose is that intent should be one of the "constituent" elements of the crime.

Sometimes drunkenness will rebut the intent necessary for an aggravated crime, but not for a simple form of the same crime. Thus if D when drunk attack P with a stick, the jury may find that he did not intend to do grievous bodily harm, so that he is acquitted of the aggravated assault, and yet he may be convicted of a common assault on the ground that even in his drunken state he did intend to do some harm. The law here works satisfactorily in saving the drunkard from conviction for the more serious crime, when in fact he lacked the essentials of it, while leaving him subject to a conviction and so open to compulsory remedial treatment.

Although no judge has hitherto said in so many words that drunkenness can negative recklessness, this is an inevitable consequence of accepted principles. Take, for example, a case of assault and battery. Suppose that D hits P on the nose. If D was sober the court may think that D intended to hit P and so was guilty of an assault and battery. But if D was drunk, and was flinging his arms about in an endeavor to stand upright, this may lead the court to conclude that the blow was not intended or forseseen. Other evidence might lead to the same conclusion, for example, that D was in an epileptic fit, or trying to swat a fly, or hailing a taxi. It is not the drunkenness that is the defense; drunkenness is no defense; it is simply a piece of evidence relevant to infer that a necessary mental state was absent.

Of course, the exempting rule would not help a man who deliberately gets drunk in order to commit a crime. He may get drunk to give himself courage to do the deed, or to muddle his senses in the hope that he will not be held responsible. If he had the requisite intent at the time he got drunk, he can be properly convicted, even if he did not have it at the time of committing the act. Such cases, however, are rare. It seems unlikely that a criminal would choose to get drunk before a crime because (1) his drunkenness may lead him to forget his purpose; (2) it will render him more clumsy in carrying out his purpose, and thus increase the risk that his intention miscarries; and (3) it will increase the risk of being found out. Although drunkenness is frequently regarded as a ground for mitigation of punishment, there is no evidence that criminals get drunk before the crime in order to take advantage of this mitigation.

Also, it is slightly misleading, though convenient, to speak of drunkenness as "rebutting" an intent. It is not strictly a rebuttal because the persuasive burden is on the prosecution. Where drunkenness goes to negative the *mens rea*, and it is disputed whether the defendant was drunk or not,

he is entitled to the benefit of any reasonable doubt.[24] But the burden is on him to introduce some evidence, although it may be slight or doubtfully relevant, to go before the jury. It is because the persuasive burden remains on the prosecution that it is misleading to speak of the accused being presumed to intend the probable consequences of his act.

Drunkenness is no defense on a charge of manslaughter because the standard of care required of a drunkard is the same as that required of anyone else. Thus if D kills P in a drunken wrath, and sets up the defense that he did not know what he was doing, the evidence may justify an acquittal of murder, but there must be a conviction of manslaughter.[25]

Drunkenness may negative knowledge or establish mistake. Thus it may help to show that the accused believed himself to be acting in self-defense, or, in rape, to show that the accused believed himself to have the woman's consent.

A misleading statement that is sometimes made is that drunkenness is a defense in crime when it causes mistake, and a mistake on the part of a drunkard may be reasonable where such a mistake on the part of a sober person would be unreasonable. The reference to reasonableness is confused because what is in issue is not the reasonableness of the accused's belief but whether the court believes the assertion that he entertained the belief. The question is whether the defense of mistake is worthy of belief, not whether the mistake was reasonable. Reasonableness of the mistake is, in crimes requiring *mens rea*, only one factor to be taken into account in treating the credibility of the defense; another is whether the accused was drunk or sober at the time. Nor is it only drunkenness that may induce one to credit that an unreasonable mistake was made; if the accused was color-blind, for example, that also might explain how he came to make an unusual mistake. It is only in crimes of negligence, like manslaughter, that the reasonableness of the mistake is in issue.

Drunken mistake, then, falls under the ordinary rules of mistake. There is no special rule for drunkenness in the law relating to mistake. Also, where the drunken mistake negatives a necessary intention of recklessness, the rule that the drunkenness is a "defense" is not a new rule, but is merely a restatement of the proposition that drunkenness may rebut a required intent. Hence the burden is on the state, and all that happens is that when all the evidence is in, the intent is held not to be established.

Drunken mistake, is, of course, no excuse for a crime of negligence. It is for this reason that one who is drunk "in charge" cannot plead that he was so drunk as not to know he was drunk, or not to know he was in charge. However, it is submitted that if he became incapacitated by drugs without negligence, he would be excused, for example, by following a doctor's orders.

ALCOHOL TESTS FOR DRUNKENNESS

The primary purpose of testing the body fluids for alcohol is to detect drunken drivers. Mere observation of an alleged drunken driver is of little value. Under the emotional stress of arrest, many who have seriously lost their normal ability to drive can temporarily assume such control of their faculties that their condition escapes detection. Also, similar symptoms to those of alcohol are produced by many other drugs which may have been taken in the treatment of disease, or for temporary relief from headache or other abnormalities. It is, therefore, imperative to accurately determine if the accused is drunk.[26]

The generally accepted standards of alcohol within the body now utilized by courts in nearly all states are those of the National Safety Council. They are based upon concentration within the blood, although any body fluid may be used with proper correction of amounts determined to be present as compared with blood. However, the results are more open to error with the majority of them than with blood and spinal fluid.

The blood standards are as follows:

0.05%—Should not be prosecuted. About two ounces of whiskey or two bottles of beer.

0.15% or less—Should not drive.

0.15% or more—Should be prosecuted as definitely drunk. About six to seven ounces of whiskey or six to seven bottles of beer.

0.2 %—Dizzy and delerious.

0.3 %—Dazed and dejected.

0.4 %—Dead drunk.

0.5 %—Dead.

With concentration of 0.15% all persons lose the clearness of intellect necessary to safely operate motor vehicles. This is true of many between 0.05 and 0.15%. So few may be unsafe drivers with alcohol blood findings of less than 0.05% that extreme doubt must be cast upon alcohol as then being a positive factor in accident production.[27]

With these standards in mind, consideration may be given to the meaning of motor vehicle laws in relation to driving while under the influence of intoxicants. Common legal phrases are "in an intoxicated condition", "in a drunken or partly drunken condition", and "under the influence of liquor". Black's Law Dictionary considers these provisions to be essentially synonymous, but not with the phrase "while intoxicated", which represents a more advanced stage of alcoholism. Black proceeds to explain that the legal provisions frequently included mean any drinking which destroys the clearness of the driver's intellect and his control of himself, mentally or physically, with impairment to appreciable degree. Having this last thought in mind, the prohibition of law presumably does not apply unless the effects of alcohol are open to observation by others than the driver who has been drinking, for the impairment must be seen; it must be present to an appreciable degree. This is not true in some people until the concentration of alcohol in the blood has reached 0.15%.[28]

Standards for the urine are somewhat higher than those for blood. The ratio of blood concentration to that of urine, according to the National Safety Council is 1.35:1. So the recommendations that legal prosecution should not follow if blood concentration is less than 0.05%, for the urine becomes about 0.04%. The conclusion that intoxication is present if 0.15% is found within the blood represents 0.11% in the urine.

Since the relationship between urinary alcohol and intoxication is more variable than for blood, it is recommended that the same standards for legal purposes be followed in the interpretation of urine. This gives the benefit of doubt due to variability to the accused. When 0.05% of alcohol is found within the urine, the blood concentration will ordinarily be about 0.07%. The finding of 0.15% urinary alcohol ordinarily discloses about 0.2% of blood alcohol. Certainly no exception can be taken by the culprit if the blood standards are applied to urine.[29]

An ounce of whiskey or a bottle of beer ordinarily leads to about 0.02% of alcohol in the blood. This may affect the higher centers of mentality, leading to a careless feeling of well being, bodily and mental comfort. The cardinal point of importance in the production of accidents lies in the fact that alcohol is a sedative to mental inhibitions. Judgment and self-control are decreased. Self criticism is blunted. At the level of one ounce of whiskey or a bottle of beer, the American Medical Association does not consider these changes to be present to sufficient degree to increase the likelihood of accident. However, when carried beyond this point in certain individuals, they may not drive safely.

From 0.05% to 0.15% alcohol within the blood has markedly variable effects within individuals. It is well recognized that some may drink much more than others. Food within the stomach usually delays absorption. Since from six to seven ounces of whiskey or bottles or beer are ordinarily required to produce 0.15% blood alcohol, the zone of questionable safety in drinking lies between one and six ounces of whiskey.

Coordination between the eye and muscular response of the hands and feet falls

below normal. There may be slurring of speech. Of prime importance as a causative factor in accident production is a feeling of detachment on the part of the individual from himself. He may observe what he does sometimes with amusement and other times with consternation at inability to perform normally. This leads many to avoid driving while intoxicated, but amusement may result in utter lack of attention and care of the consequences. At 0.15% blood alcohol, there is sufficient impairment in all individuals to seriously increase the hazard of driving cars.

Between 0.1 and 0.4% of alcohol within the blood represents the zone wherein primitive functions of mentality and motor control are seriously embarrassed. Some individuals exhibit poor coordination and perception at concentration so low as 0.1%, while in others that is not true until so high a level is reached as 0.4%. With this degree of impairment, the individual may still temporarily assume control of himself and not appear to be intoxicated. Intellect is almost gone. He responds only to strong emotional appeal, but may then control himself to a material degree. When arrested, the drunkard may temporarily appear to be sober.[30]

0.4% of alcohol within the blood is usually of extreme consequences. These people are likely to die. Sollman in his *Manual of Pharmacology* reports death as having occurred after drinking a pint of whiskey. It is well recognized that many people can drink more, but alcoholic concentrations within the body fluids or more than 0.4% are usually fatal.[31]

Considering the usual legal provision of motor vehicle laws that there must be impairment to an appreciable degree, the importance of symptoms open to observation becomes manifest. The findings of authorities parallel the standards of the National Safety Council, followed in a majority of the states, that 0.15% of alcohol within the blood leads to symptoms open to observation without exception. At this level there are found slight but definite changes in physiologic function and behavior, abnormalities visible to the critical observer, even when the accused pulls himself together under the eyes of the police. Then the accused may seem sober, but careful observation discloses that not to be true.

At between 0.18 and 0.23% the authorities report that intoxication is obvious to anyone. There is some incoordination. Speech is thick. Personality changes. Not infrequently the sense of time is disturbed. However, thought is usually rational.

At from 0.27 to 0.32% people stagger. It is difficult to remain awake. Stupor approaches.

Intoxication is more pronounced during stages of alcoholic absorption, when blood concentrations are increasing, than when decreasing. That may account to a degree for the difference reported by various observers, but the standards of the National Safety Council are sufficienlty high to avoid unjust accusation and are generally accepted by all authorities.[32]

Alcohol tends to become distributed throughout the water of the body. Thus in a short time, general equalization is to be expected, but there may well be delay. Immediately after driking, the alcohol concentration would be far higher if a test were made on the contents of the stomach than if it were made on other fluids of the body. From the alimentary tract, alcohol is absorbed into the blood stream, whose concentration is at first necessarily higher than other fluids except those of the stomach and intestines. From the blood, alcohol rapidly enters tissue fluids throughout the body, and begins to be eliminated from the lungs and kidneys. So the concentration within the brain, spinal fluid, sputum, expired air, and urine before long approaches levels permitting a test of these materials for diagnostic purposes. Urine reaches stability last, unless collected from the ureters. If there was much urine within the bladder before drinking, dilution results. Alcoholic urine may remain in the bladder long after elimination from other parts of the body has advanced to a considerable degree.

Tests may be successfully performed on the brain, spinal fluid, blood, and urine a long time after death unless embalming has

been done. That process materially destroys concentrations previously existing, because of replacement of blood by water solutions, and not infrequently embalming fluids are used to give a positive test indicative of alcohol to an extreme degree, such as formaldehyde.[33]

Many objections have been raised to standard methods of testing for alcohol within body fluids, on the grounds that other substances not infrequently present during life are registered as alcohol. This is true of acetone, produced by diabetes, but never found as a result of disease in concentrations approaching 0.15%. Paraldehyde and chloral are drugs given as sedatives to drunkards, but less than 0.01% is toxic. Acetaldehyde is found in fresh spirits, liquor which has not been aged, but concentration far below 0.15% would lead to great toxicity. Ether may have been given as an anesthetic, but the history thereof would be obvious, and concentrations of 0.05% within the blood are toxic. Chloroform may similarly be determined from the history, and less than 0.01% within the blood is toxic. People could not drive with concentrations approaching 0.15%. Carbon tetrachloride causes death at concentrations of less than 0.01% within the blood. Formaldehyde may be found in the urine of those using methanamine as a drug, but 0.01% causes death. Wood alcohol leads to drunkenness at 0.15% within the blood.

In discussing laboratory findings, the doctor may be presented with questions relative to agents that will be recorded as alcohol by ordinary testing methods, and inquiry based upon demand that they account for the laboratory conclusion of intoxication. However, consideration of the minute quantities that lead to toxicity establish the fact that the accused could not have been driving a car if under the influence of such agents at blood concentrations of 0.15%.

Extreme care must be taken that alcohol has not entered as a contaminant during the test, because of the washing of equipment therewith. The technique must be correct and equipment clean. Exact identity of the specimen of body fluid as that of the accused, with certainty that the laboratory results were derived therefrom, is imperative.

It may be argued that all organic substances can be oxidized by potassium dichromate and strong sulphuric acid, common reagents used in testing, and thus register as alcohol. However, concentrated acid is not used but dilution to less than 35%. Within the time allowed for testing, substances such as acetone, chloroform, ether, and chloral will not be oxidized and show as alcohol. The great majority of organic substances do not oxidize in time far beyond that used during the test and with acid strength utilized.

The doctor who is to interpret the laboratory findings requires an intimate knowledge of the method of testing. He must be ready to explain why other substances are not erroneously reported as alcohol. The tests are accurate to reasonable degree, and the standards followed are sufficiently high to fully warrant accusations at the levels made.

Alcohol within the brain is required to produce intoxication. So it would be most desirable that test be made of the brain or spinal fluid, whose concentration certainly will be about the same. Samples of spinal fluid ordinarily will not be given, and test of the brain is possible only after death.

Early authorities considered the brain and spinal fluid to be the only sources of value, but that is no longer believed to be true. It has been reported by the authorities that the concentrations of alcohol in these fluids are about equal to those of blood.[34]

Because of objection sometimes made to collecting even a small amount of blood, examination of the urine is frequently utilized. While early investigators declared the concentration to be about the same as for blood, it is now recognized that this is generally not true. Unless the bladder is emptied of urine present before drinking and excreted for some time thereafter, very low values will be found, through dilution. So it is recommended that a specimen be taken, tested, but others follow at half hour intervals. Not infrequently, because no

urine can be obtained during periods of emotional stress, this method cannot be utilized.

The National Safety Council has established the ratio between urine and blood as 1:1.35. It has been reputed that a very constant ratio between urine collected from the ureters at an average of 1.3 for blood is to one for urine. It was found that reasonable determination could be made if the bladder was emptied about one hour after drinking and a specimen collected one-half hour thereafter utilized.[35]

It has also been concluded that urine concentration of less than 0.07% did not disclose any considerable in capacity. Using the National Safety Council ratio of 1.35, this represents an expected concentration within the blood of about 0.09%, somewhat above the level at which prosecution is not recommended at 0.05%. The authorities found many people to be obviously under the influence of liquor with urine concentrations of alcohol between 0.07% and 0.2%. These represent blood ratios between 0.09% and 0.27%. It was concluded that with urine concentrations exceeding 0.2% all people are definitely under the influence of alcohol. That is not far different from the National Safety Council standard for legal prosecution at 0.15%.[36]

The United States Navy Laboratory Manual reports urine examination to be of little value because of the lag before alcohol appears therein, and the continuation of alcohol after intoxication has decreased, through elimination from the blood, but retention within the bladder. That is the generally accepted opinion today, and urine should be utilized only if blood cannot be obtained. It should also be recalled that error is certain should there be failure to correlate findings in the urine with standards based upon the blood. The urine concentration should be multiplied by 1.35 if blood standards as indication for intoxication are to be followed.[37]

Sputum is of interest for experimental purposes, but of little practical value during criminal studies. A sufficient amount cannot ordinarily be obtained to permit test without difficulty. The concentrations are about comparable with those of blood.

Any body fluid can be used, such as from within the abdomen when present in large quantity in ascites, from the pleura in pleurisy, and from within joints when increased following sprain. Alcohol from the blood becomes diffused throughout the water of the entire body rather rapidly, and upon elimination from the blood by the lungs and kidneys, will return thereto from all fluid collections of the body. Lag during the time of increasing blood concentration is to be expected and during elimination. Tests of such fluids are of experimental rather than practical value.

Air has been utilized considerably in the past for the detection of alcohol in the study of intoxication, because little objection may be raised by the accused of the taking of an air sample as compared with that of blood or spinal fluid, and urine may not be obtainable. Values have been determined which closely follow those for the blood at all times during increasing and decreasing body concentration. Excellent methods of determination are available: Forrester's Intoximeter, Greenberg-Keater's Alcholmeter, and the most widely used, Harger's Drunkometer.

However, many problems arise. The sample of air is ordinarily collected in a rubber balloon. It is most difficult to clean inside, and dusting powder necessary to prevent the balloon sticking together may register as alcohol during testing procedures. It is necessary to obtain air from deep within the lungs, rejecting the first expired air, which has just been inhaled, because there has not yet been opportunity for alcohol from the blood to have entered.

Early observers reported that the alcohol found in 2000 cubic centimeters was slightly less than the amount found in one cubic centimeter of urine. It was concluded that the ratio was equivalent when 1300 cubic centimeters of air and one cubic centimeter of blood was used. The difference is logical in view of the fact that the first comparison was of air to urine, and blood carries a greater concentration.

There was also developed by Dr. Harger a method of testing expired air based on the amount of carbon dioxide therein. Good

results were obtained, but variations between individuals by as much as 50% were found.

Dr. Roscoe N. Gray states in his *Attorney's Textbook of Medicine* at page 625: "It is no longer considered that alcoholic determination of air from the lungs is satisfactory." However, there is some dissent from this position by other experts who have studied and worked with this type of alcohol detection.[38]

The best fluid to be used for the alcohol test is venous blood. Capillary blood is not so satisfactory. It is difficult to obtain a sufficient amount. Contamination, as from sweat, is likely, and the flow may be so slow that a disproportionate ratio between corpuscles and serum results. Arterial blood would serve, but veins are more convenient and safer to enter than arteries. It has been asserted by the authroities than venous blood is the most practical body fluid to use at critical levels, having in mind the standard of 0.15% beyond which intoxication may be concluded as certainly present, while below that level there is doubt. Thus, if concentration approximates 0.15%, the use of other body fluids such as urine is not so conclusive. Any body fluid may be used when determination shows concentration is obviously well above this critical level, and so the accused is certainly drunk.

The ratio between blood and brain concentrations has been studied after sufficient time had passed to permit full absorption. It was determined that the brain held a higher concentration than blood in the ratio of 1.2:1. Therefore, if blood alcohol is above the level of 0.15%, leading to the conclusion that intoxication is certainly present, this drug will be found in the brain to even a greater degree. Of course, immediately after one large drink, blood concentration may exceed that of the brain, but not for long.[39]

The laboratory report sometimes does not appear in percentages, but in milligrams of alcohol per cubic centimeter of blood. Since one cubic centimeter of blood is approximately 1,000 milligrams by weight, then 1% of alcohol per cubic centimeter would be about ten milligrams. Thus, 0.01% of alcohol would be about 0.1 milligrams, and 0.05% of alcohol would be about 0.5 milligrams. Below that concentration, it is considered that no intoxication exists. The critical level at which all are to be considered as intoxicated, with 0.15% alcohol within the blood, is equivalent to about 1.5 milligrams of alcohol per cubic centimeter of blood by weight. Between 0.5 and 1.5 milligrams, certain people are sober, others intoxicated.

The modified Nicloux method of blood examination for alcohol, essentially as described in the United States Navy Laboratory Manual, is frequently followed because it does not require any color standards. These are difficult to make and spoil in time, particularly if the liquid in the color standard tubes comes in contact with the stoppers.

Technic: The reagents required are a saturated solution of chemically pure picric acid; a solution of 3.8 grams of potassium dichromate in 1,000 cubic centimeters of water; and concentrated chemically pure sulphuric acid. A flask connected to a U-shaped tube with volume markings in cubic centimeters is needed.

To make the test, 5 cubic centimeters of blood are added to 25 cubic centimeters of picric acid solution. This is shaken and allowed to stand for about 10 minutes, until brown. By then, all proteids within the blood are coagulated and precipitate to the bottom of the container.

The whole is then filtered. The filtrate is yellow in color, crystal clear. All the proteids and everything that does not remain in solution, including blood corpuscles, are removed remaining on the filter paper.

Next, 15 cubic centimeters of the filtrate is put in a flask which will stand heat, gently. To the flask is attached, by means of glass tubing through a hole in the stopper in the flask, the U-shaped tube which carries volume markings. The alcohol and some of the water is evaporated from the flask, condensing within the cold U-tube. By the time 2.5 cubic centimeters of distillate is collected, all of the alcohol will have left the blood.

Then 0.5 cubic centimeters of the distillate is placed in each of five test tubes.

Potassium dichromate solution in amount of 0.5 cubic centimeters, and 0.5 cubic centimeters of sulphuric acid is added to the first tube. These substances oxidize the alcohol, changing the color sharply. Should this remain yellow, somewhat as before adding the test substances, then there is an excessive amount of dichromate, far beyond the alcohol present. If the color becomes blue, alcohol is present in excess, beyond the dichromate. Should the color turn green, the amount of alcohol was just sufficient to unite with the dichromate added. The test tube must be permitted to stand for five minutes before final observation of the colors.

If yellow, less of the dichromate but the same 0.5 cubic centimeters of sulphuric acid is added to the next test tube. If the color is blue, then more dichromate is added. After waiting five minutes and observing whether blue, yellow, or green is obtained in the second tube, the amount is varied to be added to the third tube. So the process is continued until finally a green color is obtained.

Calculation of the amount of alcohol then proceeds as follows. The number of cubic centimeters of dichromate required to produce a green color times two, equals the milligrams of alcohol per cubic centimeter of blood. The proof of this conclusion is based upon the fact that the amount of dichromate in the test reagent is just sufficient to permit one cubic centimeter of dichromate to fully oxidize one milligram of alcohol to a green color. It may be recalled that 5 cubic centimeters of blood was used, diluted to 30 cubic centimeters. Then one-half of this was distilled, obtaining all the alcohol therefrom, and so the alcohol from 2.5 cubic centimeters of blood was in the distillate. Thus one cubic centimeter of the distillate contains all of the alcohol from one cubic centimeter of blood. Since 0.5 cubic centimeters of the distillate was placed in each of the five test tubes, the result represents the alcohol in 0.5 cubic centimeters of blood. Thus we multiply the result by two in order to determine the amount of alcohol in one cubic centimeter of blood.[40]

Urine may be studied by the same method. The first step consists of taking 5 cubic centimeters of urine and 25 cubic

centimeters of water. This is filtered. Then, 15 cubic centimeters of the filtrate is placed in a flask, just as was done in the third step for blood. From there on, the procedure is identical.

Interpretation followed by the Navy is in accordance with the same standards as the National Safety Council. Thus 0.05% or 0.5 milligrams of alcohol per cubic centimeter of blood is the top limit of those considered as in no way under the influence of alcohol to sufficient degree to warrant any prosecution before a Court Martial. If the concentration is greater than 0.15% or 1.5 milligrams per cubic centimeter of blood, intoxication is presumed to be present.

Another popular test is Bogan's Method. The reagents required are Ansties, which is ⅔ of 1% potassium dichromate in concentrated sulphuric acid. This is diluted with water in equal parts before use. Scott-Wilson reagent is 1.0 gram of mercury cyanide in 60 cubic centimeters of water, to which is added a mixture of ⅛ gram of sodium hydroxide in 60 cubic centimeters of water, and then with addition of a mixture of 0.29 grams of silver nitrate in 40 cubic centimeters of water.

Standard colors are prepared. Eleven test tubes are marked for 0, 0.5, 1.0, 1.5, 2.0, 2.5, 3.0, 3.5, 4.0, 4.5, and 5.0 milligrams of alcohol. Then 5 cubic centimeters of Ansties reagent is added to each tube. 0.05 cubic centimeters of 1% alcohol is added to the tube labelled 0.5 milligrams. This amount is increased by 0.05 cubic centimeters in each succeeding tube, thus placing in the tubes the amount of alcohol called for by the labels. Distilled water is used for the tube marked 0. Then all of the tubes are diluted with water to an equal volume. The tubes are placed in a boiling water bath for ten minutes, cooled and sealed. Variations in color appear in each tube depending on the amounts of alcohol.

Equipment required to perform the test consists of four glass tubes, each U-shaped, connected to one another by glass tubing which passes through stoppers. A water suction pump is connected to the final tube. When this is turned on, it will suck air through the entire series.

The method of testing is to place one cubic centimeter of blood or urine in the first tube, which is farthest from the water pump. One cubic centimeter of Scott-Wilson reagent is added to that tube and 5 drops of liquid petrolatum floated upon the surface to prevent foaming when air is drawn through. In the fourth tube, that closest to the suction pump, 5 cubic centimeters of Ansties reagent is placed. Nothing is put in the second and third tubes, which simply serve as traps to prevent any liquid material becoming drawn from the first into the fourth. The water pump is started, the four tubes are placed in a boiling water bath, and the air is slowly drawn through for ten minutes.

The color changes in the fourth tube. When finished, all of the alcohol has been evaporated and chemically changed in the fourth tube. Comparison with the standards will determine the amount of alcohol in one cubic centimeter of blood or urine.[41]

Alcohol Tests for Drunkenness as Evidence

Persons complaining that their constitutional rights have been invaded by the use against them, in a criminal case, of evidence secured by means of a compulsory physical examination or other invasion of their bodily integrity have most frequently relied upon the privilege against self-incrimination, or against being compelled to give testimony against oneself in a criminal case, contained in the constitutions of the United States and most of the states. The contention has met with little favor in recent proceedings in the state courts, most of which have continued to draw the distinction between "real" and "verbal" evidence, holding that the privilege protects only against "testimonial compulsion". So it has been held that the privilege was not violated by reception of evidence of blood tests,[42] urinalysis for alcohol,[43] and breath tests for intoxication in drunken driving prosecutions.[44]

Thus it is held by the courts that the accused in a criminal case suffers no deprivation of constitutional rights through the use against him of evidence obtained by means of compulsory physical examinations and tests. The privilege against self-incrimination has usually been held to relate only to "testimonial evidence", as opposed to compulsory production or surrender of real of objective evidence. However, a decision of the United States Supreme Court, without directly discrediting earlier decisions approving the use of such evidence, has held that the manner in whcih the evidence is procured is a relevant consideration in determining whether the due process requirement embodied in the fourteenth amendment has been complied with, and that compulsory physical examinations and tests may be carried out in such an atmosphere of violence and illegality as to require federal intervention to set aside state convictions secured by the use of evidence so obtained.[45]

Alcohol and Amnesia

Alcohol is a potent factor in the production of amnesia, not only when drunkenness has become so deep that the individual is unable to walk about, but with lesser amounts, automatism is common. Then the individual may seem to be a most pleasant fellow, but in an alcoholic trance, a condition that may be called pathological drunkenness. He may have no recollection of what he and others did during such a period of partial consciousness. The subject is frequently before the courts in relation to crime, and many accidents are so caused.

The drinking of even small amounts may lead to profound disturbance in thinking, with detachment of mentality. Then the individual may observe himself with amusement or abhorrance, leading to careful avoidance of circumstances that may be hazardous, such as driving cars, or to utter disregard of what may happen and with reckless abandon. It appears probable that very few people become partially unconscious to dangerous degree with less than two ounces of whiskey or two bottles of beer, but with greater dosage some develop a certain degree of automatism. With about